AN OPEN CLINICAL STUDY ON

MADHUMEGAM (DIABETES MELLITUS)

WITH THE EVALUATION OF SIDDHA DRUG

NAVAL KOTTAI CHOORANAM

The dissertation submitted by

Reg. No. 321311103

Under the Guidance of

Prof. Dr. P. PARTHIBHAN, M.D.(S)

Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the requirements
For the award of the degree of

SIDDHA MARUTHUVA PERARIGNAR

DOCTOR OF MEDICINE (SIDDHA)

BRANCH I – MARUTHUVAM

POST GRADUATE DEPARTMENT OF MARUTHUVAM

THE GOVERNMENT SIDDHA MEDICAL COLLEGE

CHENNAI – 106

OCTOBER - 2016

CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY ON MADHUMEGAM"** is a bonafide work done by **Dr. M. LATHA RANI**, Government Siddha Medical College, Chennai – 600 106 in partial fulfillment of the University rules and regulations for award of **SIDDHA MARUTHUVA PERARIGNAR** under my guidance and supervision during the academic year 2013– 2016.

Name & Signature of the Guide

Name & Signature of the Head of Department

Name & Signature of the Dean/ Principal

ACKNOWLEDGEMENT

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I first of all express my elegance to Almighty God.

I am extremely greatful to the siddhars for their blessings to me to complete this dissertation work successfully.

I extend my cordial thanks to **Prof. Dr. P. Parthibhan M.D.(S),** Joint directer,Indian medicine and homeopathy, Chennai-106, as well as my guide, for his valuable guidance, useful support and kind opinions throughout this study.

I am greatful to thank **Dr. K. Kanakavalli, M.D.(S),** Principal, Govt. Siddha Medical College, Chennai – 106, for her encouragement given during the course of this study.

I am very glad to thank **Prof. Dr. N. Anbu, M.D.(S)**, H.O.D, department of Maruthuvam, Govt. Siddha Medical College, Chennai – 106, for his valuable suggestions during my study.

I wish to express my thanks to **Dr. R. Menaka, M.D.(S)** for her useful support and constant encouragement during the course of this study.

I also extend my thanks to **Dr.U. Chithra, M.D.(S)**, for her kind opinions in this dissertation work.

I am very glad to thank **Dr. R. Sasirekha, M.D.(S),** for her kind opinions in this dissertation work.

I wish to express my sincere thanks to **DR. S. M. Chitra, M.D.(S),** for her kind opinions in this dissertation work.

I wish to thank **Dr.Vidhya M.B.B.S., D.M.R.D.,** Sonologist, Arignar Anna Govt. Hospital of Indian Medicine, Chennai-106.

I also convey my sincerethanks to **Dr. P. MURALI DHARAN, M.pharm, Ph.D**. Asst. Prof. Baid metha College of Pharmacy for doing my preclinical studies of my trial medicine.

I also convey my sincere thanks to**Mrs.S.Shakila, research officer**, central siddha research institute, Chennai-106 for doing my physico chemical analysis for my trial medicine.

I like to thank, **Prof. S. Selvaraj**, **M.Sc, M.Phil,** HOD, Department of Biochemistry, Government Siddha Medical College, Arumbakkam – 106 for my biochemical analysis.

I deeply convey my gratitude to **Dr. Sathiya Rajeswaran, M.D(S), R.O., C.C.R.S.**, Chennai-106 for his moral and timely support during my work.

I also convey my special thanks to **Dr. Manivasagam, B.S.M.S, M.Sc. Biostatistics and epidemiology,** for the part in Bio-statistical analysis of my results.

I thank Librarian **Mr.V.Dhandapani, M.Com, M.Lis,** Dr.Ambedkar Library, GSMC, Chennai-106.

I would like to thank all the teaching staffs of PG department, Govt. Siddha Medical College, Chennai – 106 for their timely suggestion and encouragement.

Last and most importantly, I am indebited to all my patients for willingly accepting themselves for this study.

Also I wish to express my thanks to my husband **Er. B.Senthil kumar** and **my children** and my well wishers for their kind co-operation.

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INTRODUCTION

Introduction

“மறுப்பது உடல் நோய் மருந்தென லாகும்

மறுப்பது உள நோய் மருந்தென லாகும்

மறுப்பது இனி நோய் வராணிரு□க

மறுப்பது சாவையும் மருந்தென லாமே”[1]

According to our ancient saints , health is a state of being well enough to function well physically ,mentally and socially.

Siddha system is not merely a compendium of therapeutics based on herbs, animals and mineral resources, it is claimed to be a philosophy of life and living. As per siddhars concept derangement of the three humours leads to diseased state. Prevention and cure are the basic aim of all systems of medicine whereas the siddha system has in addition the transcendental motivation i.e., immortality of the body using kayakalpa drugs

In India alone, the prevalence of diabetes is expected to increase from 31.7 million in 2000 to 79.4 million in 2030. The prevalence of type 2diabetes is 4-6 times higher in the urban areas as compared to rural areas. The prevalence of impaired glucose tolerance (IGT) in the rural population is also high at 7-8 %. This indicates presence of genetic basis for type 2 diabetes in ethnic Indian population[2].

The disease **“Madhumegam”** is diagnosed on the basis of **“Neerkuri”, Neikuri, Nadi Paritchai, Clinical, Pathological and Bio-Chemical Investigations.**

In Modern clinical entity – “Diabetes Mellitus” closely resembles one of the variety of **“Inippuneer”** as **“Madhumegam”**. It is a metabolic disorder

caused by absolute or relative deficiency of Insulin characterized by polyuria, Polydipsia , Polyphagia , cellular dehydration etc.

Malnutrition in childhood , obesity , truncal obesity in adults coupled with lack of physical activities ,high calorie fast food and the stress on youngsters leads to more prevalence of type 2 diabetes.

WHO firmly believes the diet, lifestyle and medicines constitutes the triangle of treatment for diseases like diabetes and simple exercise like brisk walking, swimming can help effectively in the management of the disease[3].

The entire world is facing it as an outstanding problem and the disease which not only affects the rich but also the poorest. So I think that it is the right time to conquer the disease with siddha medicines .

AIM

AND

OBJECTIVE

AIM AND OBJECTIVES

AIM

The aim of this study is to conduct a Clinical trial on madhumegam (Diabetes mellitus). To evaluate the safety and efficacy of the siddha trial drug NAVAL KOTTAI CHOORANAM on madhumegam (Type 1 diabetes mellitus).

OBJECTIVES

1. To review the Siddha literary evidences dealing with etiology, classification, signs and symptoms, diagnosis, diets, prognosis of Madhumegam in Siddha system of medicine.
2. To study Madhumegam in various literature in comparison with modern Science.
3. To understand the incidence of the disease with reference to age, sex, thinaigal, paruvakalam, socio economic conditions, diet and family history.
4. To explore and utilize, the diagnostic methods envagai thervu, mukkutram, udal thathukkal with specific reference to Naadi, Neerkuri, Neikuri.
5. To carry out a clinical trial with the siddha drug NAVAL KOTTAI CHOORANAM.
6. Modern investigations should be done to access prognosis for before and after treatment of madhumegam.
7. To systematically record the signs and symptoms and the test results of full evaluation.
8. To assess the acute and sub acute toxicity of the Siddha formulations.
9. To study the anti diabetic activity of the Siddha formulation and physicochemical analysis for safety and purity of NAVAL KOTTAI CHOORANAM.

REVIEW OF LITERATURE

SIDDHA ASPECT

REVIEW OF LITERATURE

SIDDHA ASPECTS – MADHUMEGAM

“தக்க தாரணி மானிடத்தோர்கள் கேள்
ப□க மாசல□ பமீனிருவகைழிமே
நக்க நாயகன் நாயகிக்கே சொல்
மிக்கநந்தி விளம்பிவிதித்ததே”[4]

According to therayar vagadam , “The universe consist of two essential entities that is, matter and energy which siddhars referred to as shiva and sakthi”. Shiva explained megarogam to sakthi. Here nandhi explains its symptoms to the world for the benefit of the human kind. This clearly indicates that the existence of this disease is as old as human races.

“ஆமப்பா மனிதர் செய்த கன்மத்தாலே
அரகரா மேகமென்ற ராசாவாலே”[5]

According to siddhars ,the imbalance of tridosha causes totally 4448 diseases to human beings. Among them , megarogam is considered to be the emperor of diseases.

“Á¢கினும் குறையினும் நோய்செய்யும் நூலோர்
வளி முதலா எண்ணிய மூன்று”[6]

All the disease are due to alteration of three vital humors and seven physical constituent.
The factors, which affect this equilibrium of vital humors are,

- **altered diet habit (Unavu marupadugal)**
- **Seasonal variation(kala marupadugal)**
- **Depending upon immunological status(thega vanmai)**

Madhumegam has its description in various literatures like Yugi Vaithya Chindamani, Agasthiyar Gunavagadam.

Earlier, diseases were classified only according to mukkutram. Yugi munivar classified the diseases according to cause, signs and symptoms, and also explained about the prognosis, treatment and diet, which is now followed by the modern world,he also followed the Mukkutram classification.

வேறுபெயர்கள்(Synonyms)
□குபு□
வெகுமூமீனிர□
□□ □□பு□
மேகபு□
மதுமேக□
□ரமேக□
பு□□பாடு
பு□□ளி
னிமீனி□□பு□

DEFINITION (IYAL)

It is a clinical condition characterized by frequent passage of urine more than the normal resulting in deterioration and diminution of the seven thathus.

"அண்மையாயடிக் கடிக்கு நீரிறங்கு
மடி□கடி□கு அரைநா□ த□ லே காணு□
வெண்மையான தடிய தனிற்றான் பிடிக்கும்
மிக்கான சடம் வெளுத்து மேனிகன்றும்"[7]

These lines quotes frequent micturition, more than the normal with large quantity resulting in detoriation of gradual dimnision of sevan udal thathukkal.

"நீரினைப் பெருக்கலென்று நீரிழி விலக்கணங்கேள்
நீலவாரிதி போற் குக்கி நீட்டிக்கு முரை தள்ளாகும்
நீவி கூடாது கை, கால் நீலமா வினை நேராகும்
நீள் சொனாவுரனின் மூச்சு நீசமா முயங்கக்காட்டும்"[8]

Abdomen distends like sea, slurring of speech peripheral neuritis, lassitude, dyspnoea are the symptoms of Madhumegam.

As per Athma Rakshamirrtham body becomes weak, weight loss, dryness of skin and tongue, excessive thirst, tiredness, excess sleep indicate the presence of megaroham..

VERUPEYAR – SYNONYMS:

Neerizhivu, Ennipu neer

Neerizhivu – Excess of urination[9]

Ennipu Neer – The urine is sweet in taste

NOI VARUM VAZHI-AETIOLOGY

"மேகமெனு புர□ளிவரு□□ தமீதை
விளம்புகிறேன் முன்செய்த கர்மந்தன்னால்
தாகமுடன் மதுபதார்த்தங்கள் நன்றாய்த்
தான்புசித்த லாலுஞ்சிற்றினத்தின் மங்கை
போகமதி கரித்தலா லுட்டினந்தான்
போதவே மிஞ்சுதலால் தயிர்மோர் நெய்பால்
ஏகமாய்ப் புசித்தலாற் கொழுத்த வூனை
யென்று முண்ண லுவர்நீரைக் குடித்தலாலே
ஆசையுடன் சிறுவழுதலங்காய் தன்னை
யதிகமா யுண்பதால் காலந்தப்பில்
போசனங்கள் செய்தலால் நடையலைச்சல்
போதவே யிருத்தலிரா கண்விழித்தல்
தேசமெத்கு னி□தலா □வைகளாலே
சிரந்தனிற்சூ டதிகங் கொண்டுடனே ரத்தம்
சோஷிதே யதிகமாய் மேகந்தோன்றித்
தொல்லை செய்யும் நீரழிவும் இருபதாமே."[10]

(i) Diet Habits

"கோதைய□கல□ போதை
கொழுமீத□□ றைச்ரி போதை
பாதுவாய் நெய்யும் பாலும்
பரிவுட னுண்பீ ராகில்
சோதபாண் டுருவ □□க
சு□□ல **பிரமேகந்தான்**
ஒதுபு□□ளிசேர
வண்டென வறிந்து கொள்ள"[11]

"உற்பவிக்கும் பால் நெய்யா லிறைச்சி கள்ளால்
உரிசையாய் மீன்றன்னால் வருவிருத்த
மற்பவிக்கும் பதார்த்தத் தால் மதுர வஸ்தால்
மந்தங்கள் தனிற் பொசித்தல் வேகாப் பண்டம்
குற்பவிக்குங் குளிந்த வன்ன மங்கை கோஷ்டி
குறித்த நித் திரைதவிர்த லக்கினி மந்தம்"[12]

Excessive intake of food rich in carbohydrate and fat, red meat, sweet food, raw food and sleeplessness give raise to mathumegam quotes Agathiyar and Yugi Munivar.

(ii) **Sexual Indulgence:**

"கன்னி மயக்கத்தால் கண்டிடு மேகமே"[13]

"கிரந்திப் புண்ணிரண மேகக்
கீசக னென்னுந் துன் மார்க்கன்
அருந்ததி யென்னும் பாஞ்சாலி
யன்னையைக் கண்ணுற்றானே"[14]

According to Thirumoolar and Therayar, excessive indulgence in sex causes megaroham.

(iii) Obesity:

"தற்பிவிக்குஞ் **சரீரந்தான் மிகப்ப ருக்கல்**
சல்சலந்தான் பயன்படுதல் தரிக்கும் நோயே"[15]

Obesity is one of the main cause of Madhu megam.

(iv) Psychosomatic Cause:

"இயம்பவே ஆறுகுளம் பின்னஞ் செய்தல்
ஏற்றமாய் பிராமணஸ்திரீ சங்கம் பண்ணல்
பய□பவே பால□களு□ கொ□ மீதுமீ தின்னல்
பழவை சலம் போற வழிதனைத் தடுத்தல்
அயம்பவே சூலயத்திற் சலம்விட்ட டோர்க்கும்
ஆதியாம் வேதத்தை த்தூஷிற் தோர்க்கும்
துய□பவே சூ□யனை வணத் காதா□□கு□
சுருக்கா மேகம் வந்துற்ப விக்குந்தானே"[16]

According to Yugi Vaidya Chinthamani, Megaroham may occur due to not giving proper respect to Guru, Father, Mother, Vedas and suriyangod.

V. Hereditary:

"முறைகேட்கில் ஒன்பது முயற்சியால் வந்தது
துறை கேட்கிற கருப்பத்திற் றுவங்கிய மேகங்கள்
நிறை பூத்த கொங்கையாள் நாயகன் மோகத்தால்
மறை போற்றுங் கருப்பத்தில் வளர்ந்தது மேகமே"[17]

Thirumoolar have noted in their literatures that Hereditary is one among the causes of the disease Madhumegam.Present day researchers have found out that genetic factors play an important role in Madhumegam.

vi. Excess Stimulation of Moolatharam:

ச□பான**மேகமீதா லபான வாழி**
தான் புகைக்கு மேலேறிக் கபாலச் சூடாம்
பெ□தான மேகமீதா லமீனிவெகிது
போமப்பா தசைவெந்து ரத்தம் வற்றிப்
பரிவாகித் தச வாய்வால் மந்தங் கொண்டு

பெருகி□□ மலபகித□உதான வாழி
விரிவாகித் தேகமெல்லாம் விட ரூ ராலே
மெய்யழிந்த தேகமென்ற திருபதாச்சே”[18]

Among the six Atharams the Moolatharam is situated in between rectum and genitals, just end of sacral plexus.

In the Madhumegam disease, impaired Abana vayu (excretory junction) inactivate the moola agini during that time excess intake of food causes inactivation of dhasavayu which create excessive appetite (Polyphagia) and constipation. Udanan is also affected. These changes in turn causes the derangement of seven udal thathukal.

vii.Deeds:

“தானே பூருவ விதியினால் சாரும் பிணிக ளெல்லாம்
மானேர் விழியாள் வேட்கையினால் வருந்தும் பின்னும் பசியால்
தானே பொறுத்து உண்கையினால் தாகந்தன்னால் மிகச்சோர்ந்து
தானே கமலம் புண்ணாகி செய்யும் பிரமேகச் செயல்தானே.”[19]

From the above poem, the diseases also occur as a result of bad deeds committed in previous or this birth.

MURKURIGUNAM (PREMONITORY SYMPTOMS):

Premonitory symptoms of Madhumegam are poly uria, poly phagia, poly dipsia. Madhumegam exhibits the following premonitory symptoms from its initial stage of development itself. The patient experiences voracious hunger, thirst, perspiration, exhaustion and giddiness. The excessive intake of water to quench his thirst is excreted as excessive quantity of urine (poly uria). In spite of abnormal consumption of food, stamina continues to decrease.[20]

மதுமேக□பொது கு□குண□:
கூறான **மேகமது □ருபது□கு□**
குணந்தன்னைச் சிவன்சொல்ல தேவி கேட்க
தாறான தாகமொடு சோகமேககி
தரியாமல் நீரிழித விருமல் மூச்சு
ஆறான அருரி சமீனிரி மீத□ர□மை
அடிகடிக்கடிக்குத் தண்ணீர்தான் அங்கே கேட்கல்
ஈறான இடுப்புக்குள் கடுப்பு காணல்
எலும்புழற்ற லழற்றல டெரிவு டாமே
எரிவோடு சரீரமெல்லா மறைபட்டாற்போல்
எலும்பு நோதல் நித்திரையில் லாமை
வரிவோடு மாய்விமெத் தவும்பறித்தல்
மனதுசஞ் சலப்படுதல் காற்று வேண்டல்
மொரிவோடு மேல் மூச்சு மிகவுண்டாதல்

விக்கலொடு மயக்கந்தான் மெத்தக் காணல்
தெரிவோடு தேகமெங்கும் வெளுருண்டாதல்
தேகமெத்த வாலோபப் படுதல் காணே''[21]

Common Symptoms:

Thirst	Polyuria
Poly dipsia	Cough
Anorexia	Dyspnoea
Delirium	Pain in the hip and burning sensation
Sleeplessness	loss of weight
Hiccough	Flatulance
Anaemia	Giddiness

NOI VAGAIKAL- (CLASSIFICATION)

Megarogam is classified into twenty varieties to quote from ***Agasthiar***

''உட்டிண ரோகத்தாலும் உறும்பெரும் பசியினாலுங்
கட்டவிழ் கோதை மாதர் கலவிமட்டிலா மையலாலு
முட்டறா நாலுமாறு மும் மூன்று மொன்று மொன்று
திட்டமாய் வருவதென்று திருமுனி யருளிச் செய்தார்''[22]

Yugi Munivar classifies the same as

வச□ மீத **மேகமது □ரண்டு பமீது**
வாதத்திற் பிறந்தசலம் நாலேயாகும்
பிசனித்த பித்தத்திலு ற்றபவித்த
பேராசை லகிதானு மாறு மாகு□
தேசனிந்த சேட்டுமத்திலுற்ப வித்த
□ரான சலகிதானு□ பமீதேயாகு□[23]

According to ***Theraiyar***

''கழியும் வாதம் நான்காலும் காயும் பித்த மாறாலும்
சுழியும் சேத்துமம் பத்தாலும் சொல்லும் நாலஞ்சாய்
தோன்றும்''[24]

NOI VAGAIGAL

Books	Noi enn	Vali	Azhal	Iyam
அகமீனிய□1200	20	4	6	10
□□ வைமீனிய ரிகிதாம□	20	4	6	10
தேரைய□வாகட□	20	4	6	10
தன்வந்திரி வைத்தியம்	20	4	6	10
சரபேகினிர புர□□ளிரோக ரி□ச்சை	20	4	6	10
□□ முர□ வைமீனியகா□ ய□	20	4	6	10

The above books describe twenty different kinds of megam (urinary disorders) on the basis of colour, consistency, taste, smell, weight etc.

Out of this twenty different kinds

Four varieties are caused by vali

Six varieties are caused by Azhal

Ten varieties are caused by Iyam

Madhumegam comes under the classification of Azhal.

Classification of Megam:

According to ***Yugi Vaidhya Chinthamani***

வாதநீர் வகைகள்

"தரித்திட்ட வாதத்தின் சலந்தா னாலு
தனியான நாலுக்கும் பேரே தென்னில்
அ□மீனிட்ட ஆச்ரியகெகினிமேகமீதோடு
அதன்பிறகு சுற்றமா மேகமென்று
பிரித்திட்ட பிரமிய மேகமொன்று
பேரான மாங்கரவி மேகமென்று[25]

Vali – 4

1. Neimananeer
2. Pasumana neer
3. Seezhmana neer
4. Sathaimana neer

பித்த நீர் வகைகள்

முறையான □மீத சல மாறுமாகு□
முதிர்ந்த அப்பிய மென்றும் பிரமிய மென்றும்
துறையான சாம்பீர்ணமதும்ப மென்றும்
சாத்திகமே யாறுவிதந் தன்னோ டாறு''[26]

Azhal – 6

1. Yanai kozhupu mana neer
2. Katrazhai mana neer
3. Chunna mana neer
4. Innipu megam
5. Palingu neer
6. Muyal kurithi neer

ஐயநீர் வகைகள்

''ஆறான சிலேட்பசலம் பத்து தன்னை
அரன் சொல்ல ஆத்தாள் தான் கேட்கும் போது
வாறான வசாமேக□உமீசமேக□
மச்ரியாமே கமீதோபா □த மேக□
□ றான சுரா□சு□ல முமீத மேக□
சுற்றமாம்பி னானியொட வலண மேகம்
கேறான தெழிமீதயமா மேக மென்று
செப்பினார் சிலேட் பத்தின் செலுத்துத் தானே''[27]

Iyam – 10

1. Iaya Neer
2. Thuimai Neer
3. Moolai neer
4. Ilaneer
5. Kal neer
6. Thavala Neer
7. Kazhu neer
8. Then neer
9. Uppu neer
10. Kavichi Neer

Yugi described four types under the Vatha premeham, six types under the pitha prameham and ten types under Kaba prameham. ***"Diabetes mellitus" a clinical entity in a modern medicine is closely resembles one of the type of pitha prameham ie "Madhu Megam".***

NOIKURI KUNANGAL – (CLINICAL FEATURES)

Polyurea, Polyphagia, polydipsia, perspiration, exhaustion, insomnia, giddiness and loss of weight even at normal consumption of food.

Common sign and symptoms of Pitha Prameham

"அறியவே பித்தசல மாறுக்குந்தான்
அங்கமதிற் செய்கின்ற குணத்தைக் கேளாய்
தறியவே சரீரம் வற்றி யெரிவுண்டாகும்
சடத்திலுந் நீரிலுந்தான் கவிச்சுண்டாகும்.
தெறியவே சீப்போலுங் கற்றாழை போலும்
சேல் போலுந் தேன் போலும் நாற்ற முண்டாம்
வெறியவே பீசத்திற் கோசத்திற் குதத்தில்
மிகுமீரல் நாபியிலும் வேக்கா டாமே
வேக்காடாய் விரண முண்டாய் வாய்தான் நாறும்
□ □கலொடு அருரியாயச் சுரமுண்டாகு□
தீக்காடாய்த் தேகந்தான் கிடைகொட்டாது
னிய□கமொடு மூ□ச்சைழிண்டா மய□க மாகு□
சாக்காடாய் நாவறந் தண்ணீர் தாகம்
சக்தியொடு சரீரமெல்லாந் தளர்ச்சி யாகும்
தாக்கடா மலசஞ்சலந்தான் மிகவுண்டாகும்
சமகுணந்தான் பித்த சல மாறு மாச்சே[28]

As per the above poem, polyuria, polyphagia, Polydipsia, fever, angular stomatitis, pruritis vulvae, balanoposthtis, burning sensation all over the body, loss of weight, are common signs and symptoms of pitha prameham.

MUKKUTRAIYAL:

"Vadhamai Padaithu, pitha vanniyai kathu
Seethuma Seethamai thudaithu"[29]

Vali:

Sites of Vadha:

Below Naval, Urinary bladder, intestines, Pelvis, umbilical cord, thigh, bone, skin, nerve endings, joints, Musculature, hair root.Properties :

Dryness	Lightness
Clearness	Coolness
Mobile	formless.

Function

- ❖ Praanan (uyirkaal) :

  This controls knowledge, mind and five sense organs, which are useful for breathing and digestion.

- ❖ Abaanan (Keezh nokku kaal) :

  This is responsible for all down ward movements such as passing urine, stools, semen, menstrual flow etc

- ❖ Samaanan (Nadukkaal) :

  This aids in proper digestion.

- ❖ Viyaanan (paravukaal):

  This is responsible for all movements of all parts of the body.

- ❖ Uthaanan (Mel Nokkukaal) :

  Responsible for all upward visceral movements, such as vomiting, eructation and nausea.

- ❖ Naagan :

  Responsible for opening and closing the eyes.

- ❖ Koorman :

  Responsible for vision and yawning.

- ❖ Kirukaran :

  Responsible for salivation, nasal secretion and appetite.

- ❖ Devathatthan :

  Resposible for Laziness, sleeping and anger.

- ❖ Thananjeyan :

  Produces bloating of the body after death. It escapes on the third day after death bursting out of the cranium.

In Madhumegam

Pranan	:	Normal
Abanan	:	Constipation, Noctural polyuria, frequency of micturation.
Viyanan	:	Symmetrical sensory disturbances, Peripheral neuritis, pain all over the body. Burning sensation in the sole of foot and palm, skin infection and carbuncle.
Udanan	:	Normal
Samanan	:	Poly Phagia
Nagan	:	Normal
Koorman	:	Diabetic retinopathy / Cataract
Kirukaran	:	polyphagia

Devathathan : Laziness
Thananjeyan : -

- **Azhal:**

Sites of Pitha:

Between the heart and the naval, sweat, lymph, blood, stomach, urinary bladder, heart, saliva, eye and skin.

Properties:

Dry, cold, hot, light, subtle, keen, soft, liquid, bitter.

Function

1. Anal Pittham : It promotes appetite and helps in digestion.
2. Ranjagam : It gives colour to the blood.
3. Praasagam : It gives complexion to the skin.
4. Aalosagam : It brightens the eyes.
5. Saathagam : It controls the whole body. It has the property to fulfill all the activities which the mind desires.

In Madhu megam

Anala Pitham	-	Excess hunger
Ranjaga pitham	-	Pallor sometimes
Alosagapitham	-	Dimness of vision
Saathaga pitham	-	Lassitude
Prasaga pitham	-	Dry skin

- **Iyam:**

Sites of Kapha: Above the heart, stomach, fat, sperm, tongue, uvula, bone marrow, blood, nose, nerves, bones, large intestine, eyes, joints.

Properties:

Heavy, cold, mild, watery, sweet and stable .

Function

1. Avalambagam : Lies in the lungs, controls the heart and other kabhams.
2. Kilethagam : Lies in the stomach, makes the food moist, soft and helps in digestion.
3. Pothagam : Responsible for identifying taste.
4. Tharpagam : Present in the head and responsible for the coolness of both eyes.

5. Santhigam : Responsible for lubrication and free movements of joints. It is situated in the joimts

In Madhumegam

Avalambagam	-	Normal
Tharpagam	-	Burning sensation in the eye
Santhigam	-	Joint pain
Kilethagam	-	Excessive appetite
Pothagam	-	Normal

- **Seven Udal Thathukkal (Physical constituents)**

Annamaya kosa is constituted by seven thathus.They are the basic tissues of our body.

Normal functions:

Saram:

It is responsible for the growth and development. It keeps the individual in good spirit and it nourishes the blood.

Senneer:

Blood imparts colour to the body and nourishes the muscle responsible for the ability, intellect of the individual.

Oon:

It gives shape to the body according to the requirements for the physical activity, nourishes fat.

Kozhuppu:

It helps in lubricating the different organs and maintains only matter of the body.

Enbu:

Supports the system and responsible for posture and movements of the body.

Moolai:

It fills the bony cavity, nourishes semen, imparts strength endurance and shining appearance.

Sukkilam / Suronitham:

It is responsible for reproduction. In healthy people, they function in a harmony, while in diseased people, they are deranged.

In Madhumegam:

Saaram	:	Tiredness, General weakness
Senneer	:	Pallor
Oon	:	emaciation
Kozhuppu	:	Dry skin
Enbu	:	Later stage due to infection it affects the bone and sometimes leads to amputation.
Moolai	:	Affected in Chronic stage.
Sukkilam / Suronitham:		Impotence, Sexual urge is reduced.

So, in Madhumegam, Seven Udal Thathukkal are deranged.

MUKKUTRA VERUPADUGAL-(SIDDHA PATHOLOGY):

The disease megaroham, due to external (or) internal causes affect balance in the ratio of vali, Azhal, Iyam. This imbalance affects the Keelnokkukal, which inturn affect the seven udal thathukkal. Saram gets affected and there is loss of appetite. Seeneer also get affected with the net result even if the patient eats more nourished food (polyphagia) there won't be any improvement in health.

An imbalance in pitham does imply an imbalance in other two kutrams too and causes derangement of dasa vayu and seven udal thathukkal which cause the disease and other complications.

PINIARI MURAIMAI- (DIAGNOSIS):

Diagnostic methods in Siddha system are very unique and solely based on clinical acumen of the physician.

1. **Poriyal Arithal** (or) understanding by the fire organs of perception (Mei, Vai, Kann, Mooku, Sevi)
2. **Pulanal Arithal** (or) understanding by the sense objects (Uraithal, Suvaithal, Parthal, Mugarthal, Kettal)[30].
3. **Vinadal** (or) Interrogation

Tools used by Siddha Physicians:

(1) Kanndal (Perception)

(2) Karuthal (Inference)

(3) urai (The instruction of the inspired)[31]

The application of these three is very extensive in diagnosis and treatment.

Enn Vagai Thervu (Eight tools of Diagnosis)

Naa:

Colour of the tongue, size, shape, anomalies, surface, mobility and local lesion should be noted. Coating deposition of the tongue, increased salivation, dryness of the tongue.

In Madhumegam, the tongue remains dry and at times black.

Niram:

Colour of the skin all over the body, a local region of affection, conjunctiva, tongue, nail bud, hair etc.

Vatha Udal	-	Black and whitish colour
Pitha Udal	-	Yellowish (or) Reddish colour
Kapha udal	-	White or golden colour
Thontha udal	-	Mix of two udal colours

In Madhumegam, the colour of skin is different from original complexion, discoloured.

Mozhi:

The quote from Agasthiar Vallathi

“வார்த்தையைப் பார்”

Observation of speech and voice.

In uncontrolled Madhumegam which leads to cerebrovascular disorder, speech disorder sets in.

Vizhi:

Colour, character, vision should be observed.

In uncontrolled Madhumegam cataract set in last. In longstanding cases, the Madhumegam affects retina and causes diabetic retinopathy which is the major cause of blindness.

Sparisam:

Colour of the skin (Vali, Azhal, Iyya udal), Eruption, Hemorrhages, Ulcers, Boils, trophic changes, in the skin can be identified.

Any changes in the internal organs can be noted by palpation (or) percussion.

In Madhumegam, increased tendency **for fungal infection like moniliasis and vulvities.**

In Madhumegam the skin is **dry and pale**.

Malam:

Quantity, colour, smell, froth should be observed.

In Madhumegam, **constipation sometimes yellowish loose stool are passed.**

Muthiram:

Quantity colour froth smell and specific gravity of urine should be noted.

Urine:

Colour:

In Madhumegam **clear and white.**

Specific Gravity:

In Madhumegam**, urine is thick in consistency like honey**.

Smell:

Honey like smell

Froths:

In Madhumegam the urine **is frothy at the time of urination**.

Deposits:

In Madhumegam few epithelial cells are present in urine.

Normal quantity of adult urine is 750 – 2500 ml in 24 hours.

Disturbing polyuria at night (nocturia)and Glucosuria(the presence of sugar in urine) are present.

பு□□ற□கு□:

“அருந்து மாறிரதமும் அவிரோதமதாய்
அஃகல் அலர்தல் அகாலவூன் தவிர்ந்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியே காது பெய்
தொருமுகூர்த்தக் கலைக்குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே”[32]

Collection of Sample Urine:

The patient must take well cooked food in the previous day. The intake must be proportionate to the degree of his appetite. Food intake should be taken, at appropriate time. The patient must have sound sleep on the previous night. The urine is collected on the dawn of the next day in a glass container and closed immediately to prevent contamination. This specimen must be examined with in one and half hours. This procedure should be followed strictly to get accurate observation of Neerkuri and Neikuri[33].

நெய்குறி:

"நிறக்குறிக் குரைத்த நிருமாண நீரிற்
சிறக்க வெண்ணெய்யோர் சிறுதுளி நடு விடுத்
தென்னுறத் திறந்தவெளி யேகா தமைத்ததி
னின்றதிவலை போம் நெறிவிழியறிவும்
சென்றது புகலுந் செய்தியை யுணரே" [34]

NEIKURI:

The diagnosis and prognosis of deranged Mukkutrams are studied on the basis of the behaviour of a drop of gingelly oil gently droped on the surface of the urine kept in a wide vessel in the sunlight.

"முத்தொத்து நிற்கின் மொழவதென் கபமே"[35]

In Madhumegam, the oil dropped in urine is like a pearl and if the oil spreads slowly, the prognosis of the disease is slow and good.

நாடி:

Pulse diagnosis is the confirmatory diagnosis. In Madhumegam,

இருமியே பித்தமும் வாதமுங் கூடிடில்
மருளிசல மேக□வாருனிபோலாகு□
உருவம் வேறாரு முண்டவுடன் காந்திடும்
உருகவே வூனோடு உறிஞ்சி இனிக்குமே"[36]

Coupling of Azhal and vadha naadi causes excessive urine as vast as sea, loss of weight and polyphagia .

When kabha merges with vadha ,glucosuria,emaciation ,anaemia develops.

பார்த்திடு மூன்றும் பதிந்து மெலிந்து நிற்கில்
தேர்ந்திடு மே வந் தோன்றியே பொருந்திமெய்யில்[37]

when all the three nadis ,runs in low volume,diabetes develops.

மதுமேக குண□:

"இனிக்கின்ற வாதத் திடைசேரில் ஐயந்தான்
பனிக்கின்ற கள்ளுப் பதனிபோல் நீரோடும்

கனிக்கின்ற மேனி கரைந்து வெளுப்பேறும்
தனிக்கும் மதுமேகந் தப்பாது ஐயனே''[38]

The coupling of the Vali and Iya naadi,causes increase in urination,and diabetes develops.

நீரழிவு நோயில் காணும் அவத்தைகள்:

The following complication follow gradually. If the disease is not controlled (or) left untreated.

அவமீதை – 1

''காணவே முதவலத்தைச் சரீரந் தானும்
கனமாக□பருமீனிறுகு பு□மீது வார□
வேணவே வெண்டாக்கி யகலம் பண்ணும்''[39]

Obesity sets in.
There is obstruction in urinary flow.
Urinary passage expands due to inflammation.

அவமீதை – 2

'' மிக்க இரண் டாமவத்தை விளம்பக் கேளாய்
மூணவே மூமீனி□□டைழிமாச் சு□ல
முகமழுகித் தேஜசுதான் மிகவே குன்றும்''[40]

Micturation is frequent.
Sexual desire gets.
There is also loss of complexion.

அவமீதை – 3

''நாணவே மூன்றாகு மவத்தைக் குந்தான்
நாவறளும் வாயுவது மீறுந்தானே''[41]

Tongue generally becomes dry.
Abdomen is distended due to flatulence.

அவமீதை – 4

''தானான நாலவத்தை யங்க தாகம்
சன்னியது பாத முண்டாம்''[42]

Severe thrist occurs.
Causes delirium

அவமீதை – 5

''ஐந்து வத்தைத்
தேறான பு□பெருககிதாது நஷ்ட□[43]

Quantify of urine increased.

Loss of semen (impotence).

அவமீதை – 6

“ நிலை யாறா மவத்தையுடற் கிடை கொள்ளாது
மூனான மூச்சை வரும்”[44]

Sleeplessness is present.
Difficulty in breathing is experienced.

அவமீதை – 7

“ஏழுவத்தை
மிக்கவரோ சிகஞ்சுவாசந் தேக சாட்யம்”[45]

Tongue becomes tasteless.
Difficulty in breathing is experienced .
General weakness persists.

அவமீதை – 8

“ஏனான எட்டாவ தவத்தை தானே
எழுகிரந்தி பிளவையுந்தான் மிகவுண்டாமே”[46]

Abscess is formed.
Presence of Carbuncle .

அவமீதை – 9

“உண்டாகு மொன்பதா மவத்தை கேளாய்
ஒழுங்கான ஆசாரங் கிருமி யுண்டாம்”[47]

Irregularities in daily habits like bowel habits.

Bed sore may occur.

அவமீதை – 10

“பண்டான பத்தாந்த வைத்தைக் கேளாய்
பாரமாம் சயங்கண்டு பரத்துக்கேகும்”[48]

Secondary infection like tuberculosis may sets in due to loss of immunity,
Other complication leading to death may occur.
These are the complication at 20 Madhumegam.
Other complications: Meganeer Kattigal 10.

- Madaku Katti
- Ammaiodu Kattai
- Valai Kann Katti
- Athomuga Katti
- Pei surai katti
- Kadalai katti
- Kadugu Katti

- Vithirathi Katti
- Nilapoosani katti
- Megavithirathi katti

□ரு□ □ராதவை (Prognosis)

செய்யவே வச்சரமாந் தண்ட மான
செயமான முதுகுதண்டைப் பற்றி நிற்கும்
பெய்யவே பெருநரம்பில் மேகந்தானும்
பிறக்குமென்றே தானறிந்து வாதந்தன்னால்
□ய்யவே பிறந்தகல மாறா லசாத்தியம்
பித்தத்திற் பிறந்தசல மாறும் யாப்யம்
பையவே சேட்டுமத்திற் பிறந்த பத்தும்
பரமனுரைத் தார் சாத்யம் பராபரிக்கே''[49]

''வழியும் வாதம் நான்காமே
மாறா தவிழ்தந் தன்னாலே
பொழியும் வாதம் நில்லாது
போமே மருந்தைப் பொய்யெனவே''[50]

The four types of megam caused as a result of imbalance of vali are incurable. The six types of megam arising with disparity of Azhal could be cured with great difficulty. *But **ten types of megam arising due to Iyyam are curable***.

எந்தெந்த ரோகங்களில் சிறுநீர் அதிகரித்தாலும் குறைந்தாலும் □து ?

''வெப்பு பிணியதனில் வெம்மேகத்தால் வருந்தின்
தப்பு மிகை நீரே தானிறங்கின் – செ□□□
கிராணியிற் பாண்டில் குளர்நீர் சுருங்கிற்
பிராணன் பிரியுமெனப் பேசு''[51]

Very excess of urination in Megaroham causes death.

''துதிப்பான மேகத்தில் நீரிழிவு மாகா
தோன்றிய நீரிழிவு தண்ணீர் வாதமுமாகா''[52]

If Megaroham is associated with excessive urination, it is difficult to cure. If megaroham coexist with vali it is incurable.

''மேகத்தில் நீரிழிவு மேவுமதில் வாத நோய்
வேக வயித்துள் வயிற்றுளைவு – சோக விக்கல்
பன்னு விக்கில் தன்னில் பகரிளைப்புப் பாங்க தனிற்
பின்னளை யாகாது பேசு''[53]

If in complications of Megaroham with carbuncle, Morbid thrist, excessive body heat, shock and sweat occurs and the prognosis is bad.

“வேர்வைதனிற் கபமும் மேவுமதில் விக்கல் நோய்
கார்முகில் நேர் கூந்தலாய் கண்டு மேல் – சீர்கொள்
மருத்துவத்திற்றேர்ந்த மதியுடையாராவி
தரித்திரா தென்பர்சரி”[54]

If Iya megam is associated with sweat hiccough, the prognosis is bad.

Maruthuvam:

The treatment in Siddha Medicine is aimed at keeping the three kutrams (Vali, Azhal and Iyam) in equilibrium and maintenance of the seven udal thathukal.

In siddha science, the treatment is not only for removal of the disease, but for the prevention and improving the body condition after the removal of the disease.

This is classified as

Kappu (Prevention)

Neekam (Treatment)

Niraivu (Restoration)

Kappu:

Prevention is better than cure is a proverb. Siddha principles based mainly on prevention as mentioned in “Theraiyar pini Annuga vithi” by Theraiyar.

The aim of the treatment is to bring the affected thathus and Mukkutram to normal levels by eyamma, niyamma, diet and medicine.

Neekam

For the disease Madhumegam,**NAVALKOTTAI CHOORNAM-1gm** twice a day is given .

Niraivu:

Physical, Psychological, social and economic rehabilitation of individual is known as Niraivu.

In Madhumegam, Azhal kutram and other two kutrams Vali and Iyam deranged and causes impairment of dasavayu which in turn affect the seven udal thathukkal.

MODERN ASPECT

MODERN ASPECT

Diabetes Mellitus

Definition: Diabetes Mellitus is one of the most common endocrine disorders.It is a clinical syndrome characterised by hyperglycaemia with or without glycosuria,resulting from an absolute or relative deficiency of insulin, affecting carbohydrate,protein and fat metabolism.It may be due to impairment of insulin production or its release by Beta cells of islets of Langerhans. Long standing metabolic derangement is associated with functional and structural changes in many organs especially the vascular system leading to Diabetic retinopathy, Neuropathy and Nephropathy and Atherosclerosis[55].

AETIOLOGY

"Diabetes Mellitus" was considered to be due to a lesion in the Islets of "langerhans resulting in deficient output of Insulin.Insulin hormone is secreted at pancreas, which is a compound racemose gland. The Islets of langerhans consists of the beta cells (70-80%) and alpha cells (20-30%). The islets are more numerous in the tail part of the pancreas. Beta cells secrete insulin and alpha cells secrete glucagons. Insulin is a soluble protein, first identified by Banting and Best in 1920. Insulin acts by increasing the withdrawal of glucose from the body fluids and also by reducing the addition of glucose into the body fluids. The increased withdrawal is carried out by deposition of glycogens in the liver and muscles, by enhancing the oxidation of glucose to carbondioxide and water and conversion of glucose to fatty acids (lipogenisis). It also depress neoglucogensis (Conversion of protein and fat into glucose). The most important stimulus for the release of Insulin is the blood sugar level.

Anatomy of Pancreas

The pancreas is a compound racemose gland, analogous in its structures to the salivary glands, though softer and less compactly arranged than those organs. Its secretion, the pancreatic juice, carried by the pancreatic duct to the duodenum, is an important digestive fluid. In addition the pancreas has an important internal secretion, probably elaborated by the cells of Langerhans, which is taken up by the blood stream and is concerned with sugar metabolism. It is long and irregularly prismatic in shape; its right extremity, being broad, is called the head, and is connected to the main portion of the organ,

or body, by a slight constriction, the neck; while its left extremity gradually tapers to form the tail[56].

Situation:

It is situated transversely across the posterior wall of the abdomen, at the back of the epigastric and left hypochondric regions.

Extent: Concavity of duodenum to the hilum of the spleen.

Length: Its length varies from 12.5 to 15 cm

Weight: its weight from 60 to 100 gm.

Duct System

Pancreas has a pair of ducts,

1. The Main pancreatic duct (duct of Whirsung)
2. Accessory pancreatic duct (duct of Santorini)

The main pancreatic duct is seen in the posterior part of the pancreas. It starts at the tail and runs towards the head and receiving tributaries at right angles and becoming larger in size. It joins the common bile duct to form the Ampulla of Vater, which opens in the duodenum.

Endocrine Pancreas

The endocrine part of the gland is formed by collection of cells called the Islets of Langerhan's found scattered within Pancreatic lobules,in duct epithelium and among the acini.The islets are surrounded by acinar cells. There are about 100,000 islets in the pancreas and Each islet contains 1000-3000 cells.Thus altogether there are 100-300 million(β) cells which can store 200 units of insulin and release 30-50 units of Insulin per day. Islet cell tissue is greatly concentrated in the tail and drain their secretions directly into the circulation.

A. Major cell Types:

1. **Beta (β) cells** – Comprise about 70% of islet cells and secrete Insulin, the defective response (or) deficient synthesis of which causes Diabetes Mellitus.
2. **Alpha (α) cells:**
   Comprise 20 % of islet cells and secrete Glucagon which induces Hyperglycemia.
3. **Delta (δ) Cells :**

Comprise 5 to 10 % of islet cells and secrete Somatostatin which suppresses both Insulin and Glucagon release.

4. **Pancreatic Polypeptide (PP) cells (or) F Cells:**
   Comprise 1 to 2 % of islet cells sand secrete Pancreatic Polypeptide having some gastrointestinal effects.

B. Minor Cell Types:

1. D1 Cells elaborate Vasoactive Intestinal Peptide (VIP), which induces glycogenolysis and hyperglycemia and causes secretory diarrhoea by stimulation of gastro intestinal fluid secretion.
2. **Enterochromaffin cells** - Synthesis Serotonin, which in pancreatic tumours may induce Carcinoid syndrome.

Physiology of the pancreas

1. Functions of Exocrine pancreas

The main functions of the exocrine pancreas is the alkaline secretion of digestive enzymes and proenzymes (zymogens) and prominent among enzymes are trypsin, chymotrypsin, aminopeptidases,elastase,Lactase, amylases, lipase and Phospholipase.

2. Functions of Endocrine Pancreas

The endocrine portion of the gland the Islets of Langerhans produces the hormones Insulin and Glucagon concerned with regulation of carbohydrate,protein,fat metabolism and blood sugar level.

INSULIN

1. Insulin was first isolated from the Pancreas in 1922 by Banting and Best.Insulin is a polypeptide containing two chains of amino acids linked by disulfide bridges having molecular weight of 5808.
2. It is synthesized in the endoplasmic reticulum of the β cells as Preproinsulin that give rise to Proinsulin which undergoes peptic cleavage to form Insulin and C-peptide. C-peptide connects α and β chains.
3. The half life of insulin in the circulation in humans is about 5 minutes and cleared from circulation within 10-15 minutes and stored in beta cell granules and discharged into interstitial fluid under appropriate stimulus, enters portal circulation and liver traps 50-60%

and remaining enter peripheral circulation.It is degraded by enzyme Insulinase in liver,in the kidneys and muscles.

EFFECTS OF INSULIN:

Rapid (Seconds)	Increased transport of glucose, amino acids and K+ into insulin sensitive cells.
Intermediate (Minutes)	Stimulation of Protein Synthesis
	Inhibition of protein degradation
	Activation of glycogen synthesis and glycolytic Enzymes
	Inhibition of Phosphorylase and gluconeogenic enzymes.
Delayed (hour)	Increase in mRNA for lipogenic and other enzymes.

The net effect of the hormone is storage of Carbohydrate, Protein, and fat. Therefore Insulin is appropriately called the 'hormone of abundance'. Total daily release in man is about 50 IU out of a pancreatic store of 200-250 IU of insulin**:**

WHO CLASSIFICATION:

1. Diabetes Mellitus:

(a) Insulin dependent diabetes mellitus – IDDM type I

(b) Non insulin dependent diabetes mellitus- NIDDMType II

(i) Obese

(ii) Non obese

(c) Malnutrition related diabetes mellitus – MRDM.

(d) Other types – Secondary

Hormonal

Drug induced

Pancreatic

Other abnormalities.

1. Impaired glucose tolerance – IGT
2. Gestational diabetes mellitus – GDM

		Type I	Type II
	Age	30 years	> 30 years
	Seasonal incidence	Present	Absent
	Autoimmunity	To islet cell, insulities, other autoimmune disease	Not present
	Heredity	HLA – DR3 (or) DR4 > 90%	NO HLA Connection
	Clinical	Markedly emaciated classical symptoms are present	Merkedly obese classical symptoms usually lacking.
	Serum Insulin	Less (or) Nil	May be less (or) more
	Keto acidosis	Prone	Not so
	C-Peptide	Disappears	Remains
	Treatment	By insulin	Oral hypoglycaemic drugs rarely insulin.

CLASSIFICATION OF DIABETES MELLITUS

I. Primary Diabetes Mellitus

Type - I Insulin Dependent Diabetes Mellitus -IDDM

Type - II Non-Insulin Dependent Diabetes Mellitus - NIDDM

II. Secondary Diabetes Mellitus

1. Pancreatic pathology

a) Pancreatitis

b) Haemochromatosis

c) Cystic fibrosis

d) Neoplastic disease

e) pancreatectomy

2. Excess Endogenous production of hormonal antagonists to Insulin

a) Growth hormone (Acromegaly)

b) Glucocorticoids (Cushing's Syndrome)

c) Thyroid hormone (Hyperthyroidism)

d) Catecholamines (Pheochromocytoma)

e) Pregnancy (Human Placental lactogen – Gestational DM)

f) Glucagon (Glucagonoma)

g) Counter regulatory hormones (Severe burns, trauma)

3. Iatrogenic

Cortico Steroids

Thiazide diuretics

Phenytoin

4. Liver diseases

III. Associated with Genetic syndromes

i. Lipoatrophy

ii. Muscular dystrophies

iii. Friedreich's dystrophies

iv. Down syndrome

v. Klinefelter's syndrome

vi. Turner's Syndrome

vii. DIDMOAD (ie. Diabetes insipidus, Diabetes mellitus, optic atrophy, Nerve deafness.)

PATHOGENESIS:

It is a complex affair and is still far from clear, Lack of insulin, presence of insulin antagonists, excessive neoglucogenesis, viral infection HLA system, heredity, auto immunity jointly (or) in proportion are possibly responsible for the development of diabetes.

Due to lack of insulin blood sugar level steadily rises and when it crosses the renal threshold level of 180mg/ 100cc glycosuria results. Renal threshould level, however varies with age and pregnancy. Glucose increase the osmolarity of glomerular fillrate and this results in profuse diruresis even up to 10 to 15 liters per day associated with hyponatraemia, hypokalaemia and hypomagnesaemia. Thus intense thirst, dehydration shock and crystalloid imbalance may develop.

Again as sugar is not burnt, adequately for energy requirement, fat is mobilized from the adipose tissues and large quantities of free acid circulate in the blood. Normally these are burnt in the liver. But as these are produced in large quantities of acetone which accumulate in the blood and ultimately appear in urine and breath.

Several hormones particularly the growth hormone may help in this process incompletely metabolized carbohydrate Eg. Pyruvic acid and lactic acid also accumulate in the blood. This conditions is called diabetic keto acidosis which often leads to coma and death.

Liver is enlarged due to fat infiltration and blood contains enormous amount of neutral fat and various hormones may act as insulin antagonists. Apart from there, insulin antibodies may be produced in the blood. Lastly due to continuous loss of sugar in the urine the process of neoglucogenesis from protein maybe stimulated which may result in wasting of muscles and increased urinary loss of nitrogen.

Clinical features

1. **Glucosuria:** When blood glucose level is above 180mg/dl,glucose appears in urine. It is the renal threshold for glucose.
2. **Osmotic diuresis:** The excess glucose in renal tubules decrease reabsorbtion of water result in diuresis. This leads to polyuria, polydipsia.

3. **Polyuria**: The amount of urine may be several litres in 24 hours. This is due to excessive sugar in the urine which acts as a diuretic.
4. **Polydipsia,dryness of mouth:** Polyuria decreases water content in the body stimulates taste centre and in turn increases water intake.
5. **Polyphagia and Predilection for sweet food:** This symptom is due to non utilization of sugar for energy expenditure.
6. **Asthenia:** This is due to protein depletion and increased utilization of protein for energy.
7. **Emaciation:**It is due to loss of water, glycogen and triglyceride and protein stores and gradually reduced muscle mass occurs.
8. **Pruritis vulvae**, Balanitis(genital candidiasis), Skin sepsis(boils): This is due to irritant action of sugar on the tissue and fungal (or) bacterial infections
9. **Constipation:** The stool becomes hard and bowel movement may take place after every 2 to 3 days.
10. Nausea,headache,blurring of vision
11. Mood change, irritability, difficulty in concentrating, apathy
12. Unhealed wounds
13. Frequent changes in refractive error and Cataract

Complications of Diabetes:

Virtually every tissue and organ is biochemically and structurally altered as a consequence of the hyperglycemia of diabetes.Two biochemical mechanisms appear to be involved in the development of many complication. In the first glucose reversibly bind to the body proteins. This is a non enzymatic event that can cause structural and functional abnormalities of the involved proteins. The concentration of glycosylated hemoglobin in the blood is now used clinically as a measure of therapeutic control.

The second biochemical mechanism operate in the lens of the eye, Kidney and peripheral nerves. These tissues are endowed with an enzyme, aldose reductase, that facilitates the accumulation of sorbital and fructose in cells of the hyperglycemic patient. As a result of the intracellular accumulation of sorbital and fructose an osmotic gradient is established and excessive amounts of water enter the cells from the extra cellular compartment. The cells then swell and are damaged.

ACUTE COMPLICATIONS:

Hypoglycaemia:

Hypoglycaemia, defined as a blood glucose concentration of less the 2.5 mmol/L, occurs commonly in diabetic patients treated with insulin and infrequently in those taking a sulphonylurea drugs. Severe hypoglycaemia result in severe morbidity ie coma, convulsion, brain damage, stroke, myocardial ischaemia, vitreous haemorrhage, hypothermia and accidents.

Symptoms of Hypoglycaemia:

Sweating, trembling, hunger, anxiety, confusion, drowsiness, speech difficulty, nausea, tiredness and headache.

DIABETIC KETOACIDOSIS:

As a result of polyuria there will be dehydration, hyponatremia and hypopotassemia. The intra cellular water comes out in the extra cellular space as a result of hyperosmolarity of the extracellular fluid due to increase of glucose. Plasma volume is decreased, blood pressure falls and renal blood flow is diminished resulting in oliguria. The cellular glycogens and protein are catabolised.

Sugar is not burnt, fat is metablised from the adipose tissues and large quantities of free fatty acid circulate in the blood. Normally these are burnt in the liver, into carbondioxide and water. But as these free fatty acid are produced in large quantities.Acetyl to enzyme – A – accumulates and after condensation forms acetoacetic acid and its derivatives beta hydroxybutyric acid and acetone, which accumulate in the blood and ultimately appear in urine and breath. Incompletely metabolised carbohydrate. Eg pyruvic acid and lactic acid also accumulate in the blood. This conditions is called diabetic ketoacidosis.

When ketone bodies accumulate the cells are further dehydrated ultimately ph of the blood falls. This acidosis stimulates the respiratory centre where by pulmonary ventilation is increased giving rise to air hunger. The breath contains the smell of acetone which is very diagnostic of this condition. The function of the brain cell is depressed and gradually coma supervenes.

The cardinal biochemical features of diabetic ketoacidosis are :

- Hyper glycaemia
- Hyperketonaemia
- Metabolic Acidosis

Diabetes and the eye

Cataract

The cataract is a clouding of the lens of the eye. Which causes the vision to become blurred or dim because light control pass through the clouded lens to the back of the eye. There is a very common eye condition that develops as people get older but some one with diabetes may develop cataract at an earlier age than someone without diabetes.

Diabetic Retinopathy

Diabetic retinopathy is the most common cause of blindness in adults between 30 and 65 years of age in developed countries.

Clinical features of Diabetic Retinopathy

Micro aneurysms
Haemorrhages
Hard exudates
Soft exudates
Intra retinal microvascular abnormalities
Neovascularisation
Venous Changes
Fibrosis

Diabetic Neuropathy

This is a relatively early and common complication affecting approximately 30% of diabetic patients. Although in a few patients it can cause severe disability it is symptomless in the majority.

Clinical Features

Symmetrical sensory polyneuropathy
Asymmetrical motor diabetic neuropathy
Mononeuropathy

Autonomic neuropathy

Diabetic neuropathy usually progresses through several stages.

Diabetic foot

The foot is a frequent site for complications in patients with diabetes and for this reason foot care is particularly important.

Clinical features of Diabetic foot

Neuropathy	**Ischaemia**
Symptoms	
• Paraesthesiae	* Claudication
• Pain	* Rest pain
• Numbness	

A GUIDE TO FOOT CARE[57]

Do's

- Inspecting one's own feet daily or have someone else to inspect them for any breaks in the skin, blisters, calluses or corns.
- Washing feet daily in luke warm water. To avoid extremes of temperature and always checking the temperature of the water before using it.
- Using moisturising cream for dry fissured skin and apply powder, especially between the toes, if the skin is prone to moistness.
- To have feet measured carefully when purchasing shoes and to buy lace-up shoes with plenty of toe room.
- Inspecting the shoes before wearing them, both inside and outside for foreign objects.
- Keeping feet away from heaters, hot water bottles and other hot objects.
- The feet should be checked everytime while visiting the Clinic.

Dont's

- Not to use tobacco in any form
- Avoiding unaccustomed lengthy walks, for example, when on holiday.
- Not to cut nails very close to the edge and not to have sharp edges.

- Should not try to be one's own doctor, since corns and calluses have to be treated with professional help only.
- Not to use over-the-counter (OTC) Medicines. Rather to consult doctor.
- Not to walk barefoot, even at home
- Avoiding ill fitting shoes.
- Not to wear socks with tight elastic, soft corns socks are best.
- Avoiding hard-sole footwear
- Not to wear shoes with dividers between the toes.

DIETARY MANAGEMENT

General Principles

Proportion of Energy

Energy	**Recommended Diet**
Carbohydrate	50 - 55%
Fat	30 - 35%
Proteins	10 - 15%

Types of Diabetic Diet

Two basic types of diet are used in the treatment of diabetes:

Low-energy weight - Reducing Diet

Weight Maintenance Diet

Management of Diabetes mellitus

The aim of treatment is to achieve normal blood glucose levels,to alleviate symptoms and to prevent complications.

The four pillars of Diabetic management are,

1. Diet
2. Exercise
3. Drugs –Oral hypoglycaemic agents and Insulin therapy by regular monitoring of glycaemic control.
4. Early detection and treatment of complications.

A. Diet It is the cornerstone of management of Diabetes.The objective is to have good glycaemic control and to provide a nutritious and balanced diet. In type 2 diabetes the calories need to be restricted in order to avoid obesity.

B. Total caloric intake:

It depends on body weight,degree of physical activity and presence of comorbid illness.

Body mass index: It determines the total caloric requirement

BMI= Weight(in kg) / Height in m^2.

BMI Normal range: 22- 25.

The calories are derived from three principal sources like Carbohydrates,Proteins and Fats.

Carbohydrates

The amount of carbohydrate recommended in the diet is upto 50-60%. Whole grains, Ragi, Wheat, Millets, Oats, Brown rice which have low glycaemic index are recommended.

Proteins

Recommended amount is12-20% of total calorie intake. Dhals or Grams with outer skin and Sprouts,lean meat ,fish, egg white and chicken are preferred.

Salt - Dietary salt should be less than 6g/day.

Fat

It should be 20-24% of total intake. Sunfloweroil, Gingely oil,Safflower oil,Oive oil rich in Mono and Polyunsaturated fats are advised. Palmoil, Coconut oil and Vanaspathi should be avoided.

Milk and milk products:

Contribute to 40-45% of total fat content of vegetarian diet. Skimmed milk Unsweetened Yogurt, Curd, Buttermilk are recommended.

Vegetables: Fibre rich greens, brinjal, cauliflower, gourds and salads are advised.

Diet Plan for Diabetes

Time	Regime
Morning 6.00	Coffe/tea/ skimmed milk without sugar 1 cup
Morning 8.30	Idli-2/dosa-2/chapatti-2/pongal 1 cup
Morning 11.00	Vegetable soup/lemon juice/buttermilk 1 cup/ any fruit
Afternoon 1.00	Rice-2 cups/chapatti-3/dhal/watery vegetables Chicken/fish/butter milk
Evening 4.00	Moong dal/Channadal/coffee/tea
Evening 6.00	any fruit
Night	Idli-2/chapathi-2/rice-1 cup
Before bedtime	Milk 1 cup without sugar

Physical activity

Exercise forms an important component along with drugs and diet management in type 2 DM.Patients should be encouraged to take regular physical activity in form of Walking, Jogging, Swimming, Gardening and Cycling for 30 minutes daily. This improves Insulin sensitivity, prevents complications of Diabetes, and assist in maintaining Lipid profile and Bloodpressure, improves muscle strength and beneficial for mental state of individual.

TRIAL DRUG

TRIAL DRUG

PREPARATION OF TRIAL MEDICINE

Drug Name :Naval kottai chooranam

Text reference:

Aringar Anna Government Hospital of Indian Medicine Pharmacopeia (pgno. 11)[58]

Preparation of Naval kottai Chooranam:

Ingredients :

- Syzygium cumini
- Emblica officinalis
- Cassia fistula
- Gymnema sylvestre
- Cassia auriculata
- Curcuma longa
- Cyperus rotundus
- Terminalia chebula

Procedure :

The herbs present in naval kottai chooranam are shade dried and powdered.

Dosage : 1 – 2 gm/ bd

Adjuant : Water

Duration : 90 Days

LITERATURE REVIEW OF TRIAL MEDICINE

Naval kottaiChooranam

- **Botanical name :Cyperus rotundus**

  Suvai: thuvarppu

  Thanmai: thatpam

  Pirivu: inippu

அதிசாரம்பித்தம்அனற்றாகம்ஐயங்

குதிவாதஞ்சோபங்கொடிய-முதிர்வாந்தி[59]

- **Botanical name :Emblica officinale**

  Suvai: thuvarppu, inippu,pulippu

  Thanmai: thatpam

  Pirivu: inippu

ஒடுமிதைச்சித்தத்தில்உன்னஅனலுடனே

கூடுபிறமேகமும்போங்கூறு[60].

* Daily intake of Emblica officinale juice with fresh bitter gourd juice will stimulate the pancreas to secrete insulin[61]

- **Botanical name : Gymnema sylvestre**

  Suvai: kaipu

  Thanmai: veppam

  Pirivu: karpu

அக்கரங்கள்தீர்க்கும்அதுசுரந்தாகந்தொலைக்குந்

தக்கசிறுகுறிஞ்சாந்தான்[62]

* Water extract of gymnema sylvestre leaf tested in alloxan induced rat showed reduction in blood glucose level[63].

❖ **Botanical name : Terminalia chebula**

Suvai: thuvarppu

Thanmai: thatpam

Pirivu: inippu

* It is Glucosidase inhibitor- Retard the liberation of D-Glucose from dietary complex and delay glucose absorption.

❖ **Botanical name : Syzygium cumini**

Suvai: thuvarppu

Thanmai: thatpam

Pirivu: karppu

மாந்தம்விளையும்வலிகரப்பானுண்டாகும்

சேர்ந்ததொருநீரிழிவுஞ்சேருமோ[64]

* 4g/kg dose level found to exhibit maximum hypoglycemic effect in rabbits,3 hrs after medication.[65]

❖ **Botanical name : Cassia auriculata**

Suvai: thuvarppu

Thanmai: thatpam

Pirivu: inippu

மோகத்தினாலேவிளைந்தசலம்வெட்டையனல்

ஆகத்தின்புண்ணோடருங்கிராணி–போகத்தான்

ஆவரைபஞ்சங்கொள்அத்திசுரம்தாகமும்போம்

ஏவாரைக்கண்டமடமாதே![66]

Antidiabetic activity of various extracts of cassia auriculata on alloxan induced diabetic rats has potent anti diabetic and anti oxidant property.

❖ **Botanical name : Cassia fistula**

Suvai: thuvarppu

Thanmai: veppam

Pirivu: karppu

Aqueous extract of Cassia fistula increases insulin secretion and reduce blood glucose levels in Streptozotocin induced diabetic rats[67].

❖ **Botanical name : Curcuma longa**

Suvai: karppu, kaipu

Thanmai: veppam

Pirivu: karppu

தலைவலிநீரேரற்றஞ்சளையாதமேகம்

உலைவுதருபீனிசத்தினூடே[68]

Aqueous extract of Curcuma longa increases insulin secretion and reduce blood glucose levels in Streptozotocin induced diabetic rat .

TRIAL DRUG-NAVAL KOTTAI CHOORANAM

NAVAL KOTTAI

KONDRAI POO

KADUKAI THOL

SIRU KURINJAN ILAI

AAVARAM POO

MANJAL KIZHANGU

NELLI VATRAL

KORAI KIZHANGU

NAVAL KOTTAI CHOORANAM

NAVAL KOTTAI CHOORANAM

MATERIALS AND METHODS

MATERIALS AND METHODS

Introduction

The clinical trial for **Madhumegam(Diabetes Mellitus**) was decided to be conduct as an open label study.

Study place

The entire study was conducted on patients attending OPD of Govt Siddha Medical College,Chennai attached to Arignar Anna govt hospital for Indian medicine, Arumbakkam,Chennai-106, during the period of 2014-2016.

Data collection: Literary evidence from various,

- Siddha books
- Modern books
- Medical journals
- Internet

Population:

The Population consists of Diabetes accompanied by Polyuria Polyphagia,Polydipsia, generalised tiredness,Fatigue,Peripheral neuritis, Itching all over the body and satisfying the inclusion and exclusion criteria mentioned below.

Sample size: 40 patients.

Inclusion criteria:

1. Subjects within 3^{rd} to 6^{th} decade.
2. Non-Insulin dependent diabetes mellitus
3. Polyuria, polyphagia, polydipsia, nocturia
4. Noticeable weight loss
5. Fatigue, lethargy, tiredness
6. Peripheral neuritis,
7. Pruritis all over the body

Criteria for Exclusion(based on clinical history)

1. History of Insulin dependent diabetes mellitus
2. History of Secondary diabetes
3. History of Cardiovascular diseases
4. History of Diabetic nephropathy
5. History of Diabetic retinopathy
6. Vulnerable population such as Pregnant women,Lactatingmothers,TB affected individuals, HIV positive (Clinical History)

Duration of treatment: 90 days

Patients were followed under the guidance and supervision of the HOD, Professor,Reader,Lecturer and Asst.Lecturer of the Pothu Maruthuvam, P.G.Department, GSMC, Chennai-106.

The patients were carefully studied for their history, clinical examinations, investigations and management.

Evaluation of Clinical Parameters

The history includes past, personal, family, occupation, dietary habits and associated history.

Clinical Investigations:

Blood

TC (cells/cu mm)
DC (%)
ESR (mm)
Hb (gms%)
Sugar (Random, Fasting, Postprandial)
HbA_1C
Blood urea
Serum cholesterol

Urine

Albumin
Sugar
Deposits

Siddha Assessments:

Envagaithervugal

Neerkuri

Neikkuri

A case sheet format was prepared based on the Siddha methodology example **Envagaithervugal, Mukkutram, Nilam, Kaalam, Udalthathukkal including Neerkuri and Neikuri.** Individual case sheet was maintained for each patient at outpatient department.

RESULTS

AND

OBSERVATIONS

RESULTS AND OBSERVATIONS

The study on **Madhumegam** was carried out in 40 patients in the Outpatient department PG of Pothu Maruthuvam,Govt Siddha Medical College attached to Arignar Anna Govt hospital for Indian medicine,Chennai-106, during the period 2012-2014 were analysed.The observation were made and tabulated with following criteria.

1. Age distribution
2. Sex distribution
3. Occupational status
4. Socio economic status
5. Dietary habits
6. Family history
7. Kaalam
8. Paruva Kaalam
9. Thinai
10. Duration of illness
11. Mukkutram

a. Vali
b. Azhal
c. Iyam

12. Ezhu udalthathukkal
13. Ennvagai thervugal
14. Naadi
15. Neikuri
16. Clinical features
17. Clinical prognosis
18. Urine sugar

a. Fasting, b.Postprandial

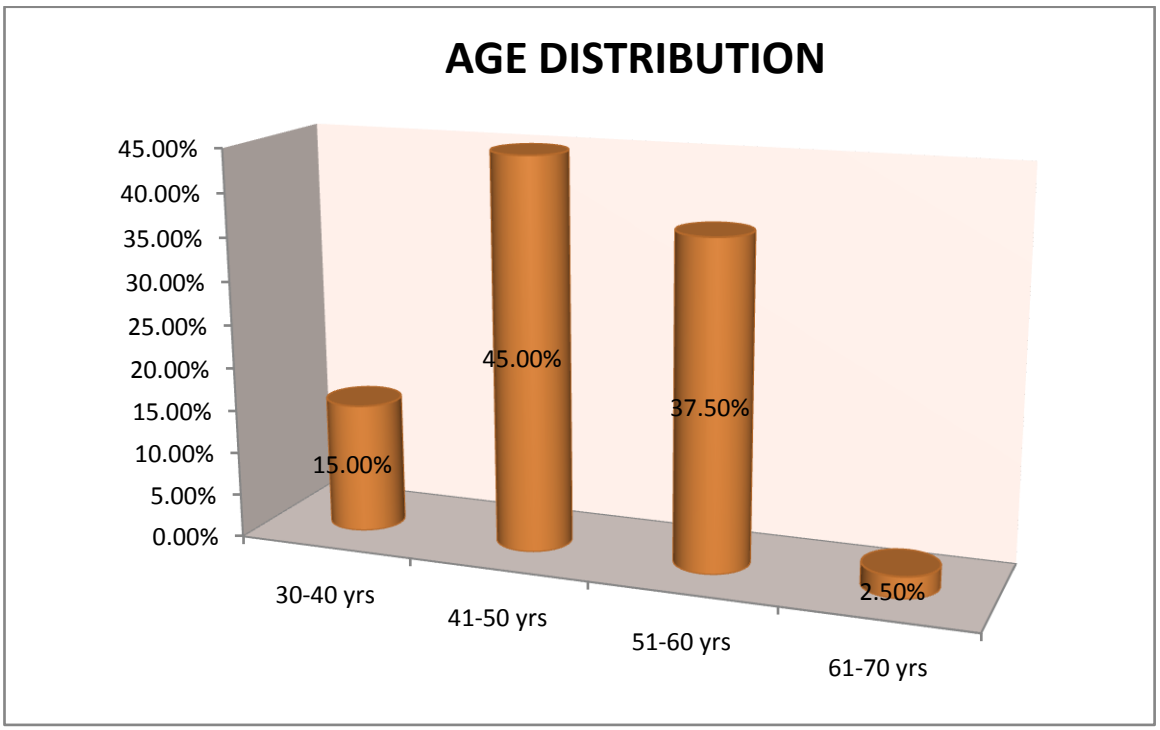
19. Blood sugar

a. Fasting, b.Post prandial

20. HbA_1C Level
21. Grading of results

1. AGE DISTRIBUTION

S. No	Age groups	No. Of cases	Percentage (%)
1.	30- 40 years	6	15%
2.	41- 50 years	18	45%
3.	51- 60 years	15	37.5%
4.	61- 70 years	1	2.5%

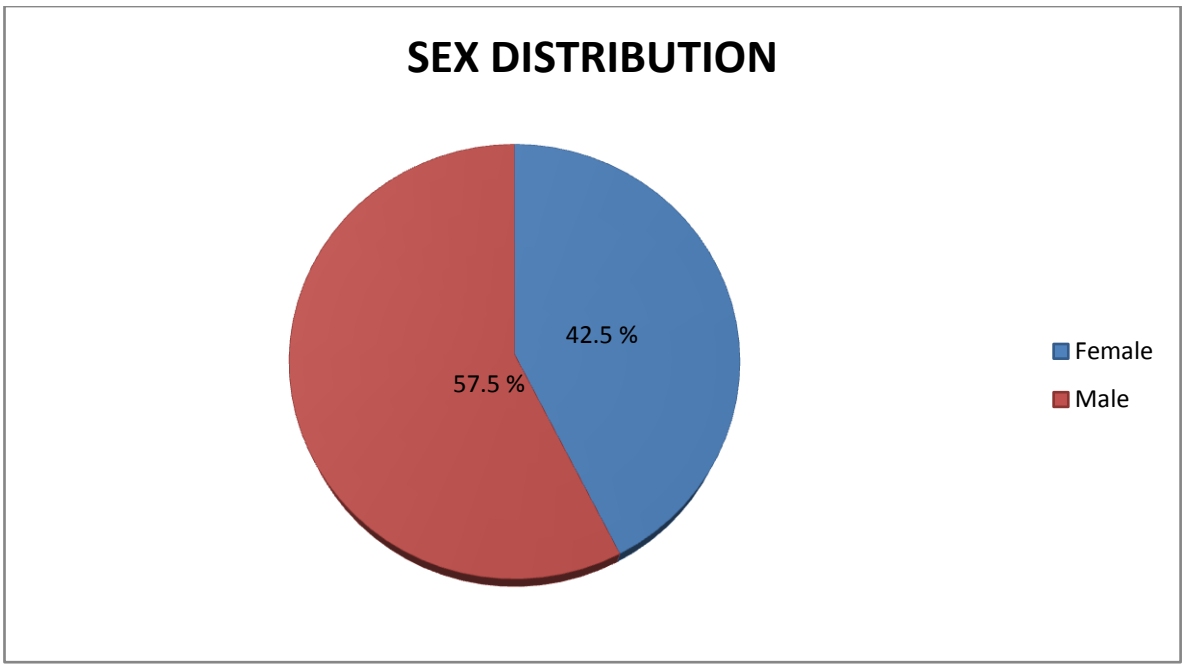


Inference:

From Selected 40 cases,6 patients (15%) were between 30-40yrs, 18 patients (45%) were between 41-50, 15 patients (37.5%) were between 51-60, 1 patient (2.5%) were between 61-70 yrs.

2. SEX DISTRIBUTION

S. No.	Sex	No. Of cases	Percentage (%)
1	Male	23	57.5%
2	Female	17	42.5%

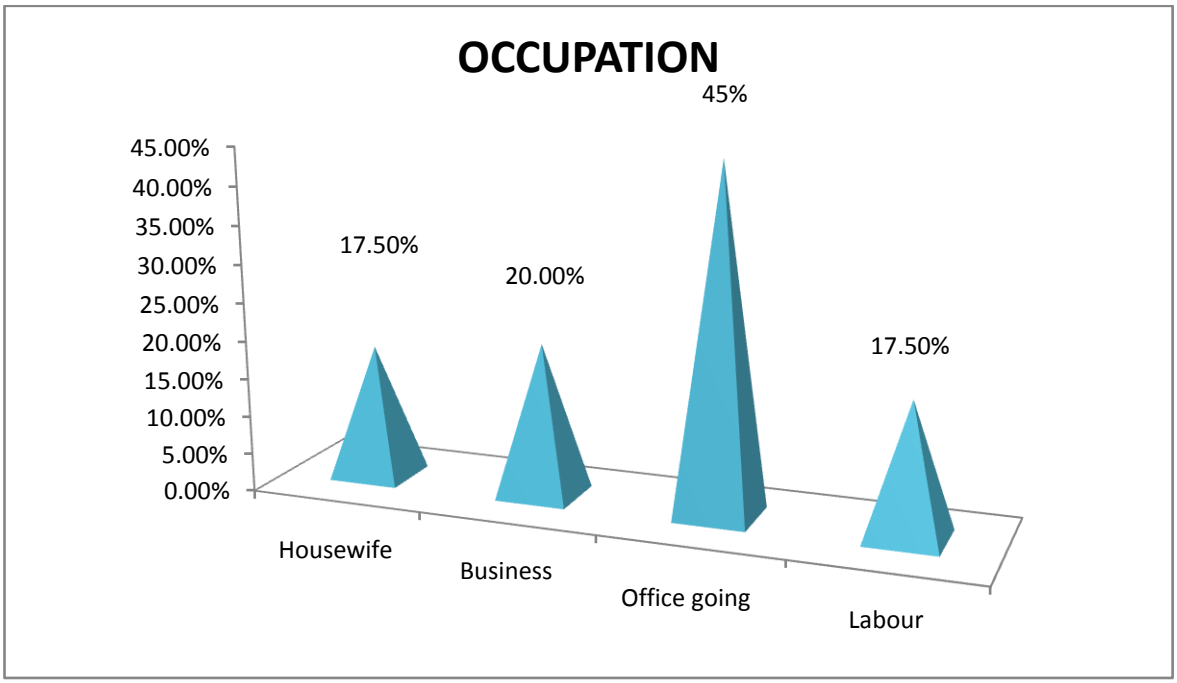


Inference:

Out of 40 patients, 17 cases (42.5%) were female and 23 cases (57.5%) were male.

3. OCCUPATIONAL STATUS

OCCUPATION	NO. OF CASES	PERCENTAGE
House wife	7	17.5%
Business	8	20%
Office going	18	45%
Labour	7	17.5%

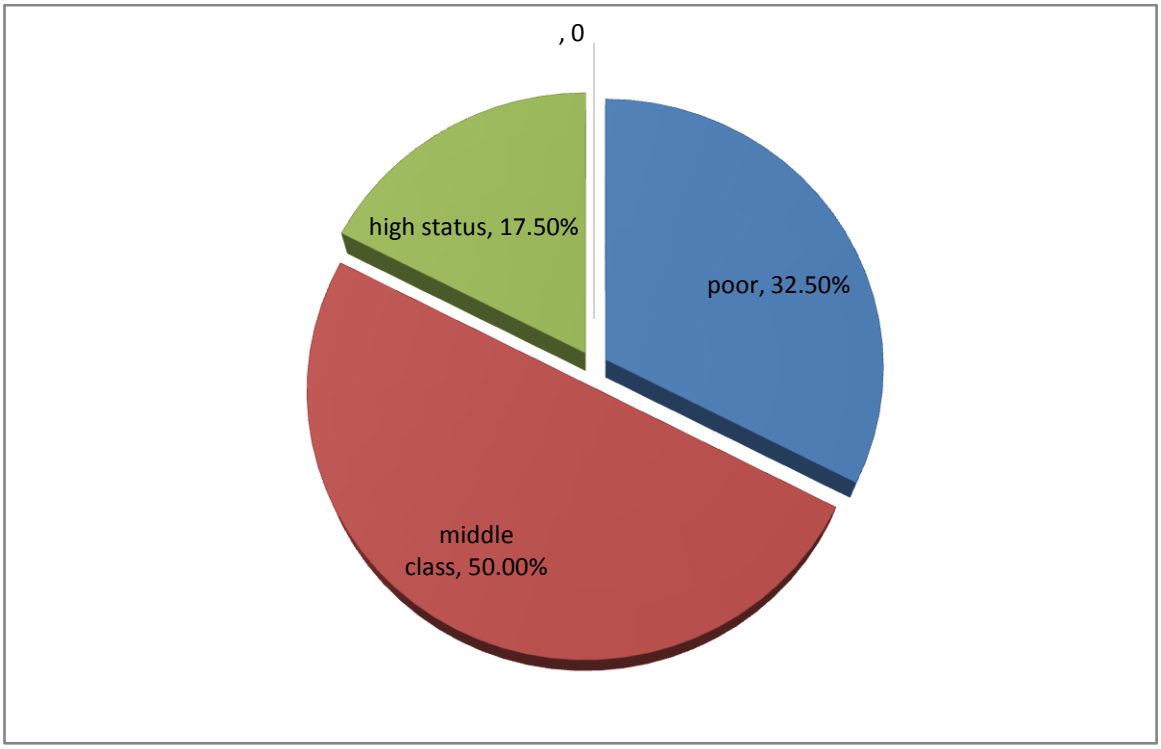


Inference:

Out of 40 patients, 7 patients (17.5%) were housewife, 8 patients (20%) were doing business , 18 patients (45%) were office goers, 7 patients (17.5%) were labourers.

4. SOCIO-ECONMIC STATUS

S. No	Socio-economic status/Month	No. of cases	Percentage (%)
1.	Poor (below 20,000)	13	32.5%
2.	Middle class (20,000- 50,000)	20	50%
3.	High status (Above 50,000)	7	17.5%

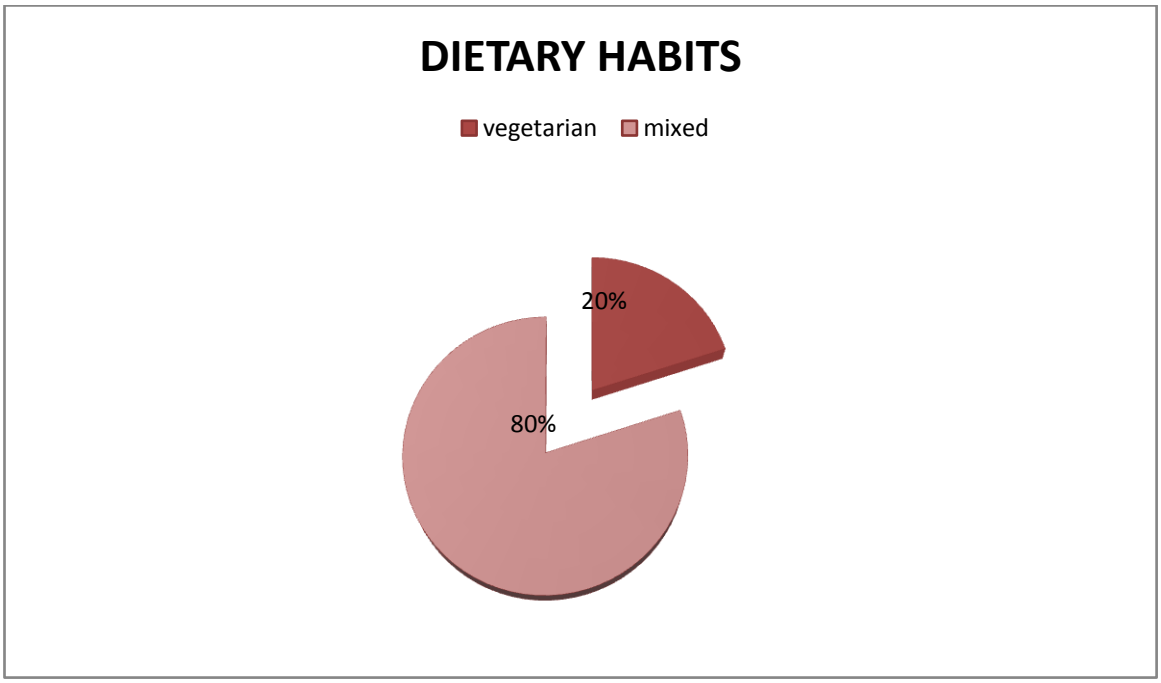


Inference:

Regarding socio-economic status, 13 patients (32.5%) comes under poor category, 20 patients (50%) comes under middle class category, and 7 patients (17.5%) comes under high class category.

5. DIETARY HABITS

DIETARY HABITS	NO. OF CASES	PERCENTAGE
Vegetarian	8	20%
Mixed	32	80%

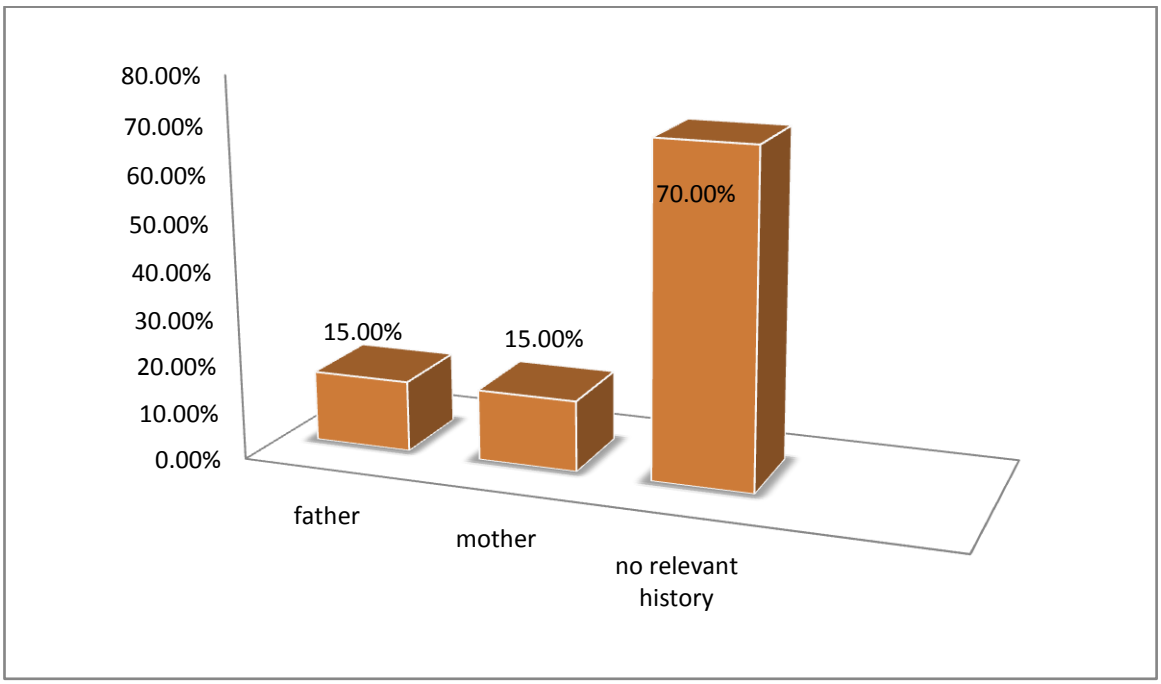


Inference:

Regarding Diet, out of 40 patients, 8 patients (20 %) take vegetarian diet and 32 patients(80%) takes mixed diet.

6. FAMILY HISTORY

FAMILY HISTORY	NO. OF CASES	PERCENTAGE %
Father	6	15%
Mother	6	15%
No Relevant History	28	70%

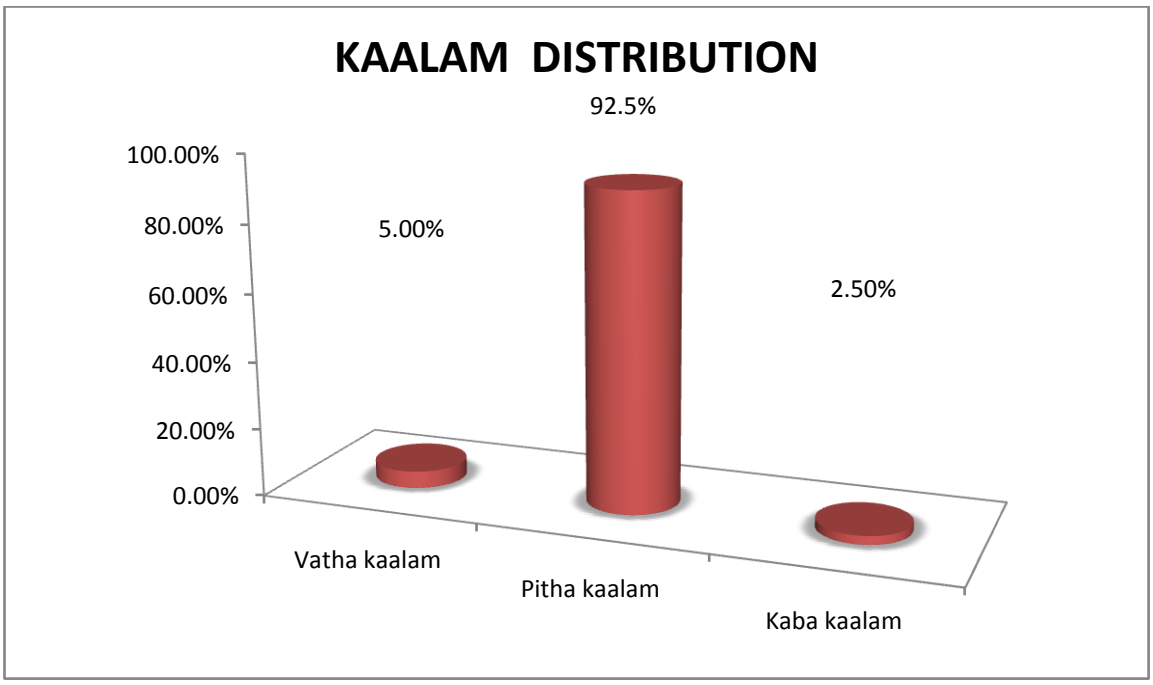


Inference:

Regarding Family history, 6 patient's(15%) father was diabetic, 6 patient's (15%) mother was diabetic and 28 patients (70%) had no family history of Diabetes.

7. DISTRIBUTION OF KAALAM

KALAM	NO. OF CASES	PERCENTAGE
Vatha Kaalam 0- 33 years	2	5%
Pitha Kaalam 34-66 years	37	92.5%
Kaba Kaalam 67-100 years	1	2.5%

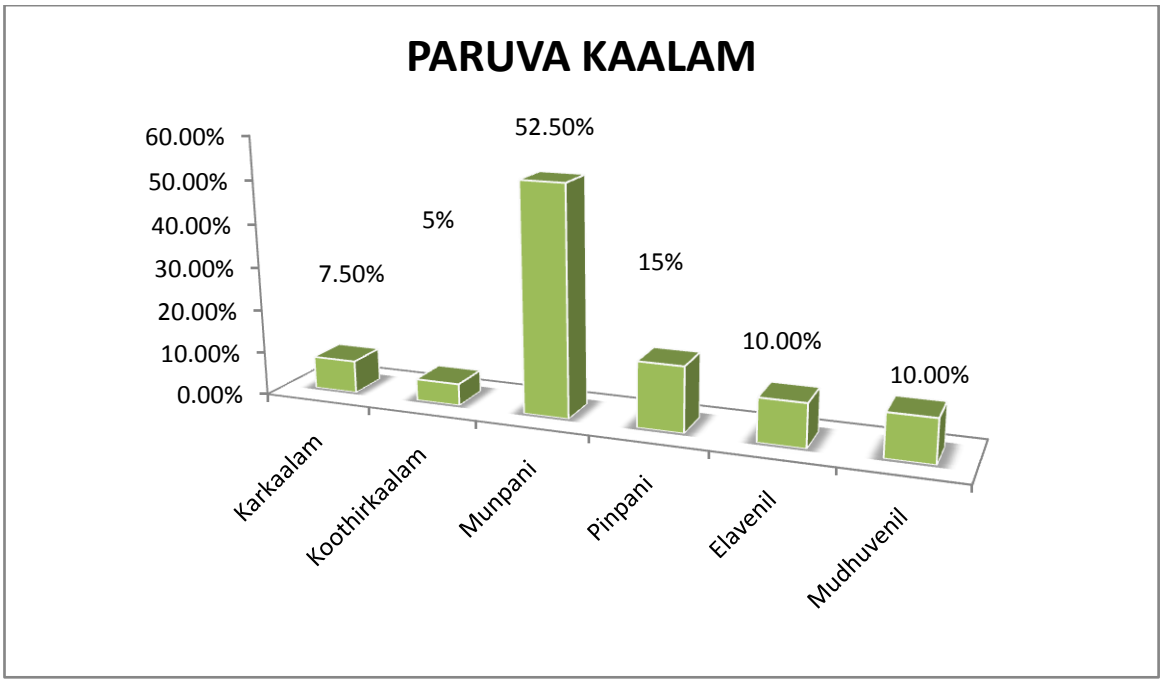


Inference:

Out of 40 patients, 2 patients (5%) comes under Vatha kaalam, 37 patients (92.5%) comes under Pitha kaalam, and 1 patient (2.5%) comes under Kaba kaalam.

8.PARUVAKAALAM

S.No	Paruvakaalam (Seasons)	Month	No.of cases	Percentage(%)
1.	Kaarkaalam	Avani,Puratasi(Mid Aug-Mid Oct)	3	7.5%
2.	Koothirkaalam	Iyppasi,Karthigai (Mid Oct-Mid Dec)	2	5%
3.	Munpani	Margazhi,Thai (Mid Dec-Mid Feb)	21	52.5%
4.	Pinpani	Maasi,Panguni (Mid Feb-Mid April)	6	15%
5.	Elavenil	Chithirai,Vaigasi (Mid April-Mid June)	4	10%
6.	Mudhuvenil	Aani,Aadi (Mid June-Mid Aug)	4	10%

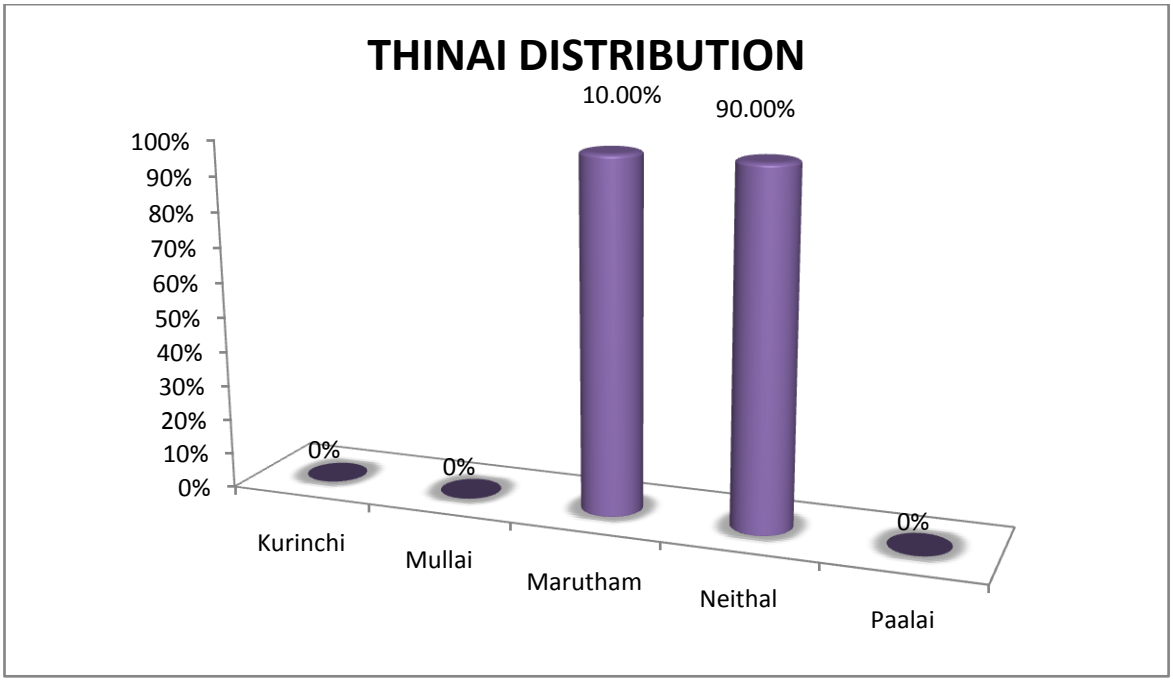


Inference:

From selected 40 patients, 3 patients (7.5%) comes under Kaarkaalam, 2 patients (2.5%) comes under Koothir kaalam, 21 patient (52.5%) comes under Munpani, 6 Patients (15%) comes under Pinpani,4 patients comes under Elavenil and 4 patients comes under Mudhuvenil .

9. DISTRIBUTION OF THINAI

THINAI	NO. OF CASES	PERCENTAGE
Kurinji	0	0
Mullai	0	0
Marutham	4	10%
Neithal	36	90%
Paalai	0	0

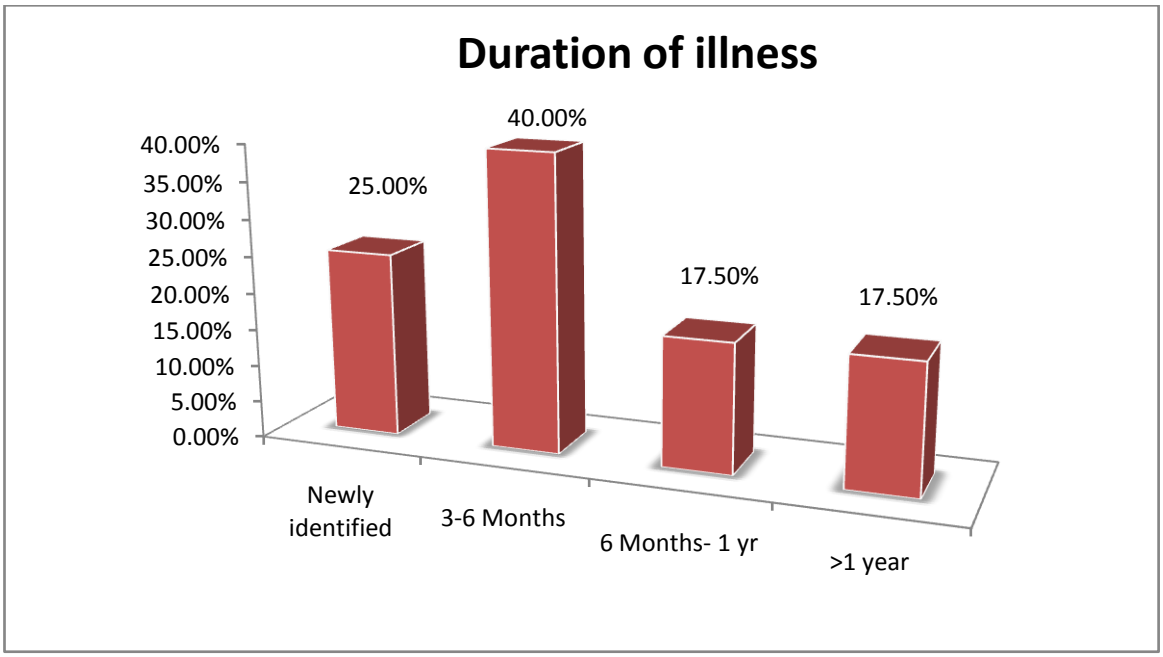


Inference:

Out of 40 patients, 4 patients (10%) comes under Marutham, 36 patients (90%) comes under Neithal nilam.

10. DURATION OF ILLNESS

S. No	Duration of illness	No. of cases	Percentage (%)
1.	Newly identified	10	25%
2.	3-6 months	16	40%
3.	6 months to one year	7	17.5%
4.	More than one year	7	17.5%



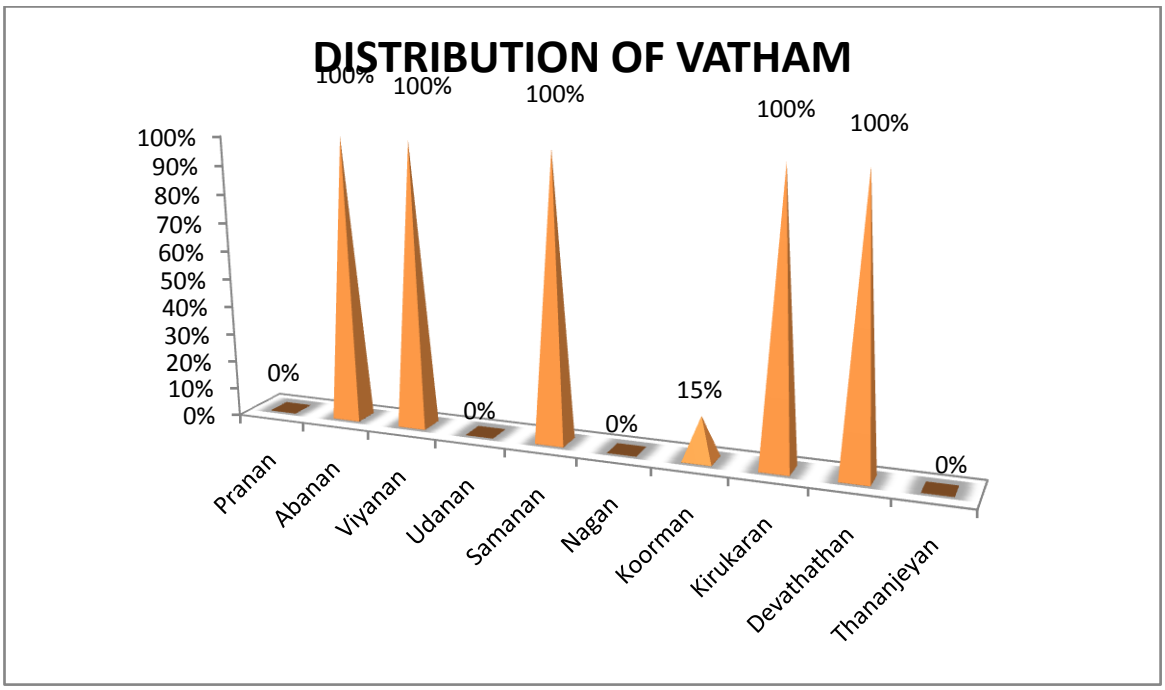
Inference:

Out of 40 patients,10 patients (25%) belongs to newly identified category, and 16 patients (40%) belongs to 3-6 months category, 7 patients (17.5%) belongs to 6 months to one year category, 7 patients (17.5%) belongs to more than one year category.

11.REFERENCE TO MUKKUTRAM

a. Affected vali:

S. No	Classification of Vali	No. of cases	Percentage (%)
1.	Pranan	0	0%
2.	Abanan	40	100%
3.	Viyanan	40	100%
4.	Udhanan	0	0%
5.	Samanan	40	100%
6.	Naagan	0	0%
7.	Koorman	6	15%
8.	Kirugaran	40	100%
9.	Devathathan	40	100%
10.	Dhananjayan	0	-

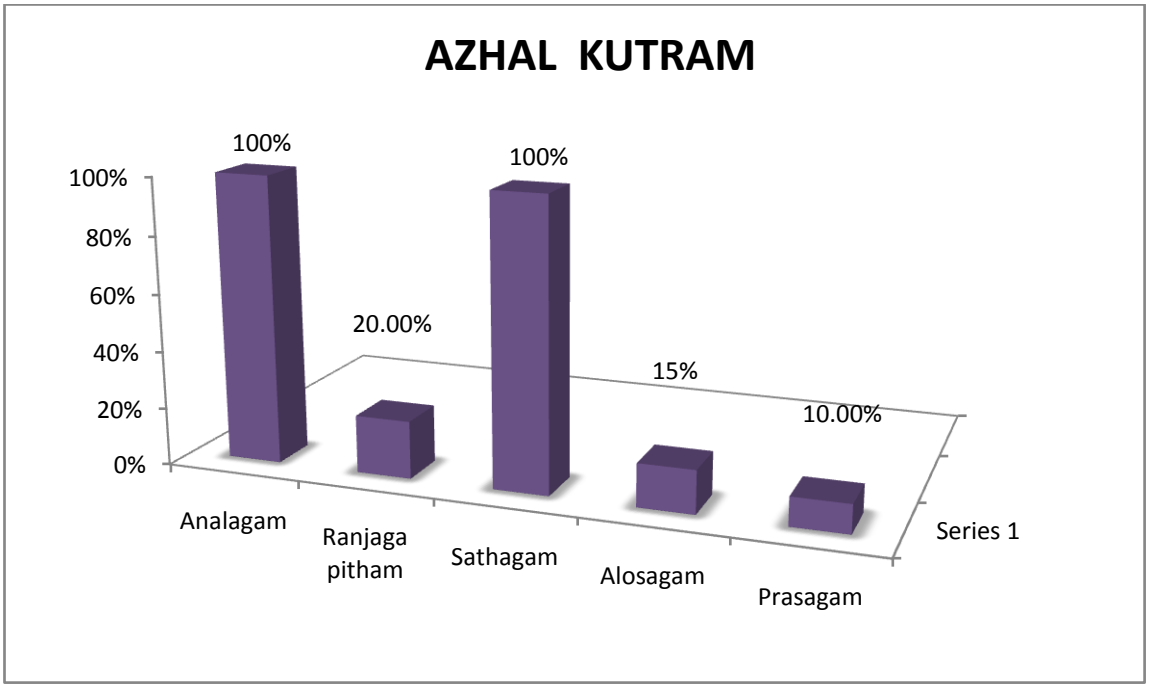


Inference:

From the Selected 40 patients, Abanan was affected in 40 patients (100%), Viyanan was affected in 40 patients (100%), Samanan in 40 patients (100%), Koorman in 6 patients (15%), Kirugaran in 40 patients (100%), Devathathan in 40 patients (100%).

b. Affected azhal

S. No	Classification of Azhal	No. of cases	Percentage (%)
1.	Analagam	40	100%
2.	Ranjagam	8	20%
3.	Saathagam	40	100%
4.	Aalosagam	6	15%
5.	Prasagam	4	10%

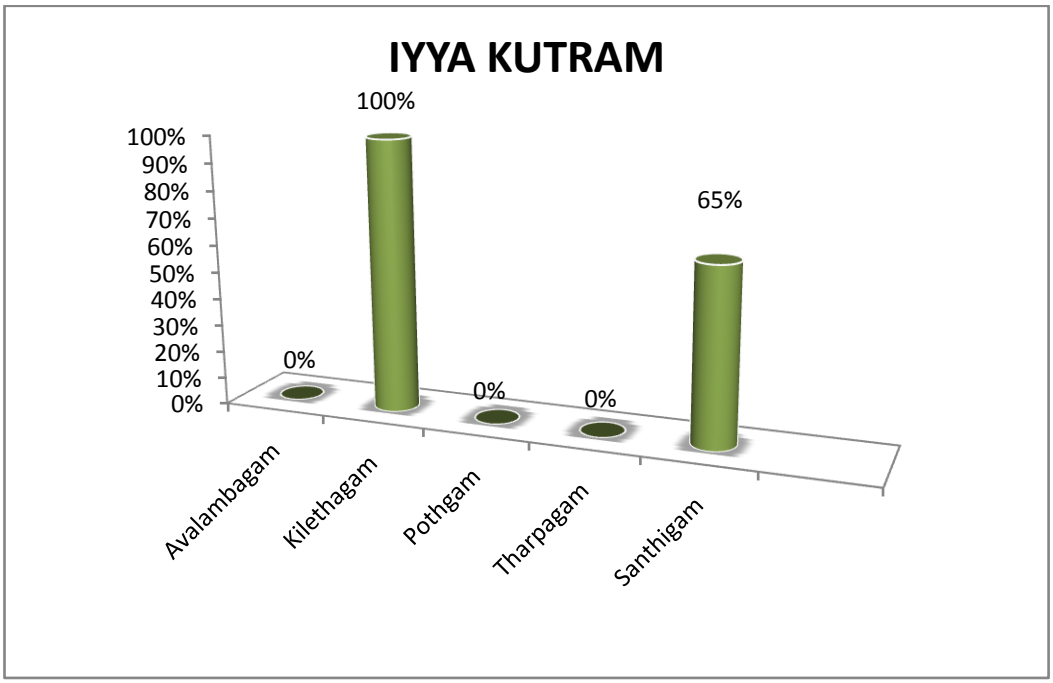


Inference:

Out of 40 patients Analagam and Sathagam affected in all patients (100%), Ranjagam affected in 8 patients (20%), Aalosagam was affected in 6 patients (15%) and Prasagam was affected in 4 patients(10%).

c. Affected iyyam

S. No	Classification of Iyyam	No. of cases	Percentage (%)
1.	Avalambagam	0	0%
2.	Klethagam	40	100%
3.	Pothagam	0	0%
4.	Tharpagam	0	0%
5.	Santhigam	26	65%

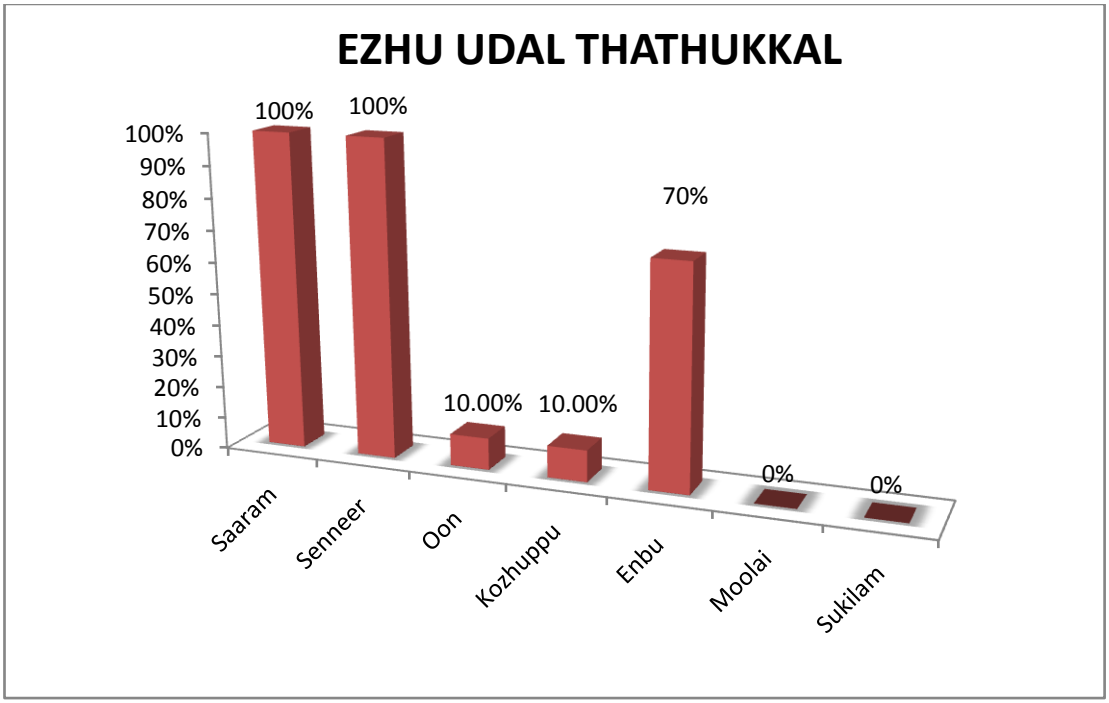


Inference:

Out of 40 patients, Kilethagam was affected in 40 patients (100%), Santhigam was affected in 26 patients (65%).

12. EZHU UDAL THATHUKKAL

S. No	Ezhu udal thathukkal	No. of cases	Percentage (%)
1.	Saaram	40	100%
2.	Senneer	40	100%
3.	Oon	4	10%
4.	Kozhuppu	4	10%
5.	Enbu	28	70%
6.	Moolai	0	0%
7.	Sukkilam/Suronitham	0	0%

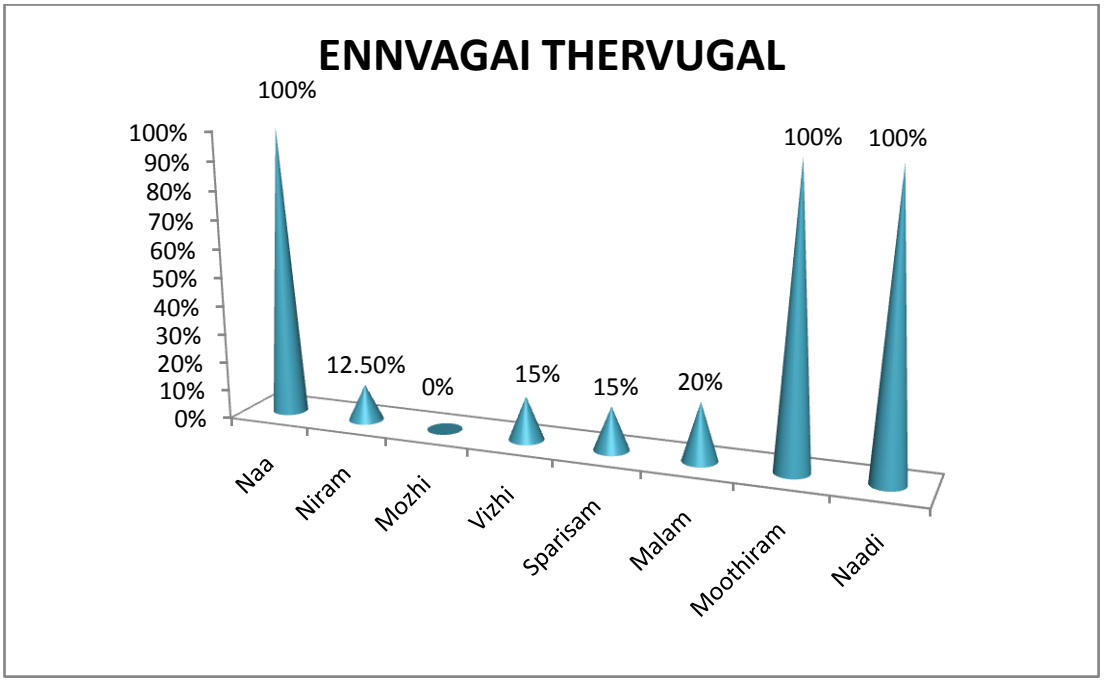


Inference:

From the above chart we can observe that Saram and Senner were affected in all patients ie.100%. Oon, Kozhuppu affected to the extent of 10% and 10% respectively and enbu affected in 28 patients(70%).

13. ENVAGAI THERVUGAL

S. No	Envagai Thervugal	No. Of cases	Percentage (%)
1.	Naa	40	100%
2.	Niram	5	12.5%
3.	Mozhi	0	0%
4.	Vizhi	6	15%
5.	Sparisam	6	15%
6.	Malam	8	20%
7.	Moothiram	40	100%
8.	Naadi	40	100%

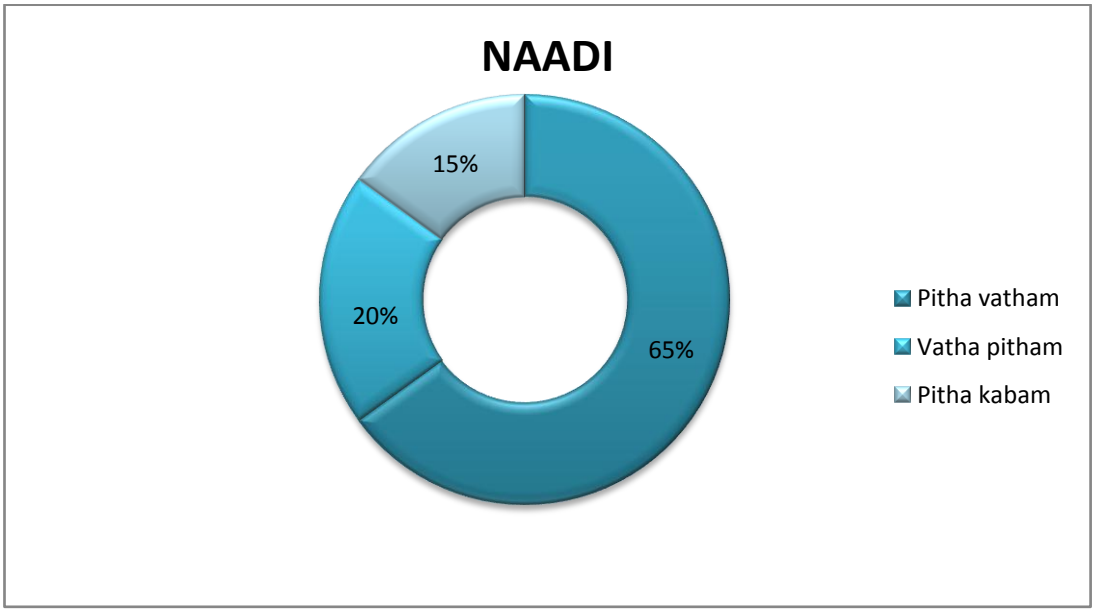


Inference:

Regarding Envagai thervu, Naa was affected in 40 patients (100%), Niram was affected in 5 patients (12.5%),Vizhi was affected in 6 patients (15%),Sparisam affected in 6 patients(15%) and Malam was affected in 8 patients (20%) and Moothiram in 40 patients(100%), Naadi affected in 40 patients(100%).

14. NAADI

S. No	Naadi	No. Of cases	Percentage (%)
1.	Pitha vatham	26	65%
2.	Vatha pitham	8	20%
3.	Pitha kabam	6	15%

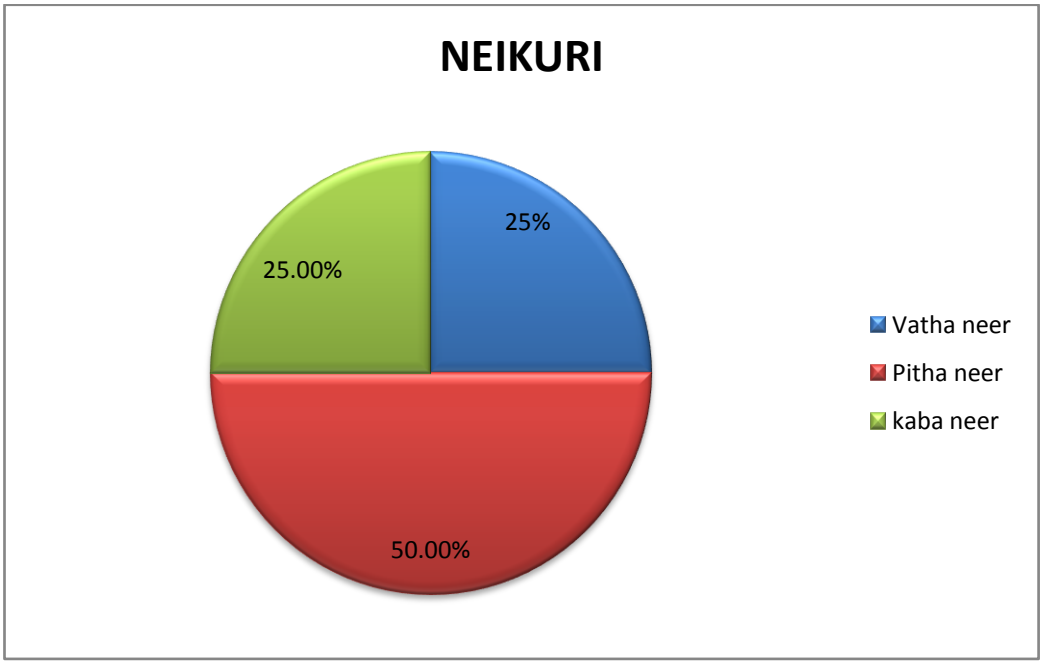


Inference:

26 patients(70%) had PithaVatha Naadi and 8 patients(20%) had Vatha Pitha naadi and 6 patients(15%) had Pitha kaba naadi.

15. NEIKURI REFERENCE

S. No	Neikuri	Character of urine	No. of cases	Percentage (%)
1.	Vatha neer	Spreads like snake	10	25%
2.	Pitha neer	Spreads like ring	20	50%
3.	Kabha neer	Float like pearl	10	25%

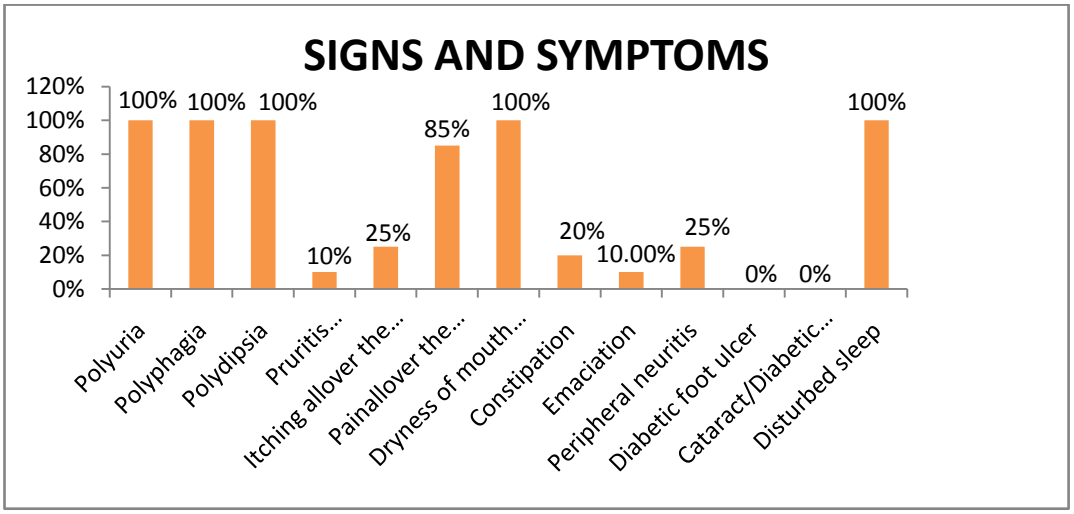


Inference:

10 patients (25%) had vatha neer, 20 patients (50%) had Pitha neer,10 patients (25%) had Kabha neer.

16. CLINICAL FEATURES

No	Signs& Symptoms	No. of cases	Percentage (%)
1.	Polyuria	40	100%
2.	Polyphagia	40	100%
3.	Polydipsia	40	100%
4.	Pruritis vulvae/Balanitis	4	10%
5.	Itching all over the body	10	25%
6.	Pain all over the body	34	85%
7.	Dryness of mouth and throat	40	100%
8.	Constipation	8	20%
9.	Emaciation	4	10%
10.	Peripheral neuritis	10	25%
11.	Diabetic foot ulcer	0	0%
12.	Cataract/Diabetic retinopathy	0	0%
13.	Disturbed sleep	40	100%

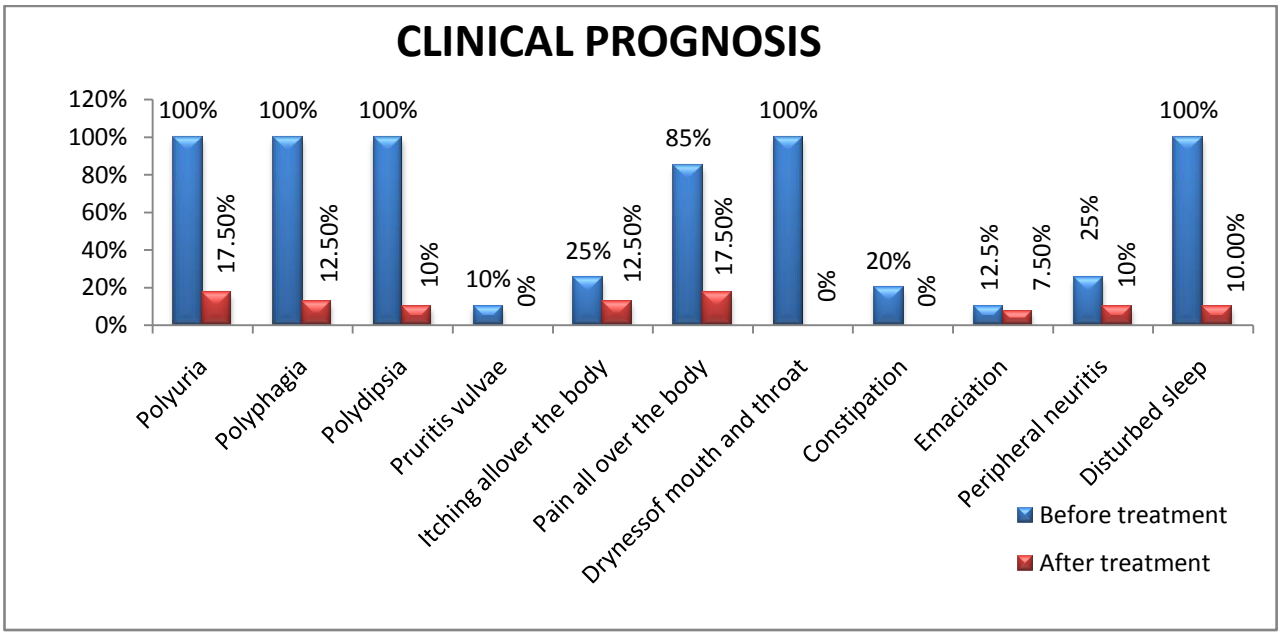


Inference:

In respect of the patients with Madhumegam the clinical symptoms of Polyuria, Polyphagia, Polydipsia, dryness of the mouth and throat and disturbed sleep were present in all cases i.e 100%.Pruritis vulvae present in 4 cases(10%),Itching all over the body in 10patients(25%),Pain all over the body in 34cases(85%),Constipation in 8 patients (20%),emaciation in 4 cases(10%) and Peripheral neuritis in 10 cases (25%).

17. CLINICAL PROGNOSIS

No	Signs&Symptoms	Before Treatment		After Treatment	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Polyuria	40	100%	7	17.5%
2.	Polyphagia	40	100%	5	12.5%
3.	Polydipsia	40	100%	4	10%
4.	Pruritis vulvae	4	10%	0	0%
5.	Itching all over the body	10	25%	0	0%
6.	Pain all over the body	34	85%	7	17.5%
7.	Dryness of mouth and throat	40	100%	0	0%
8.	Constipation	8	20%	0	0%
9.	Emaciation	4	10%	3	7.5%
10.	Peripheral neuritis	10	25%	4	10%
11.	Disturbed sleep	40	100%	4	10%

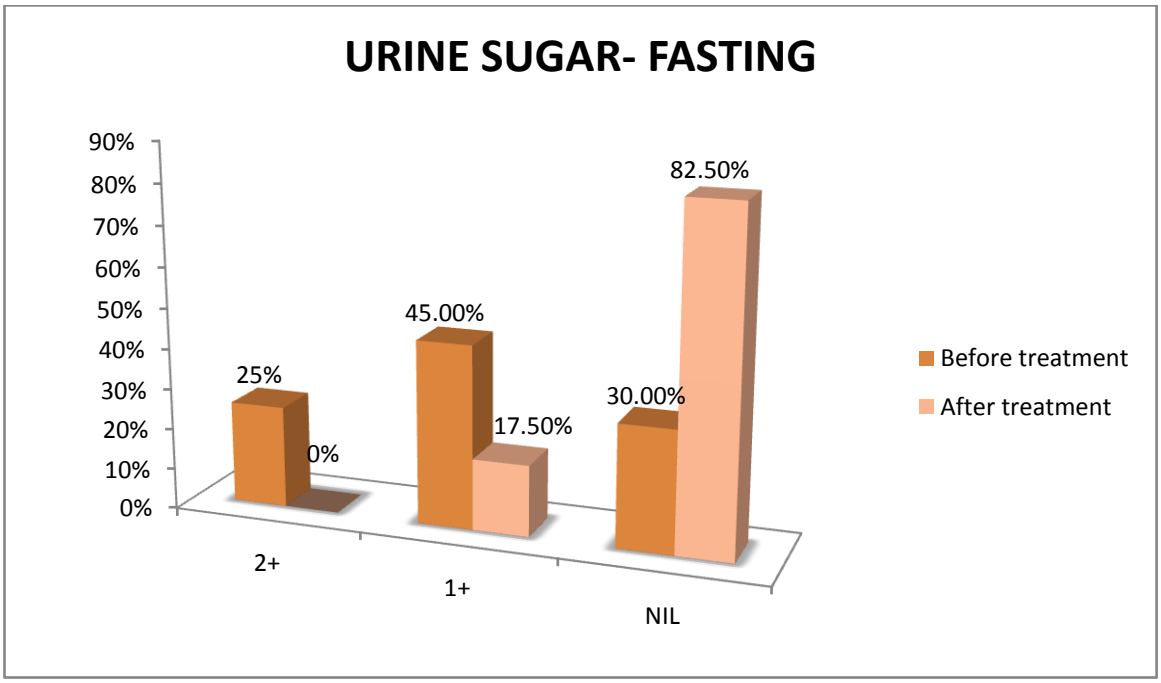


Inference:

The clinical signs and symptoms were improved after treatment showing only 17.5% of the people have poly urea, 12.5% of the people have polyphagia,10% of the people have polydipsia , 17.5% had pain all over the body, 10% have peripheral neuritis and 10% have disturbed sleep. The symptoms of Pruritis vulvae,itching all over the body,dryness of mouth and constipation were completely relieved.

18.(A) URINE SUGAR - FASTING

URINE SUGAR	BEFORE TREATMENT	PERCENTAGE %	AFTER TREATMENT	PERCENTAGE%
++	10	25%	0	0%
+	18	45%	7	17.5%
NIL	12	30%	33	82.5%

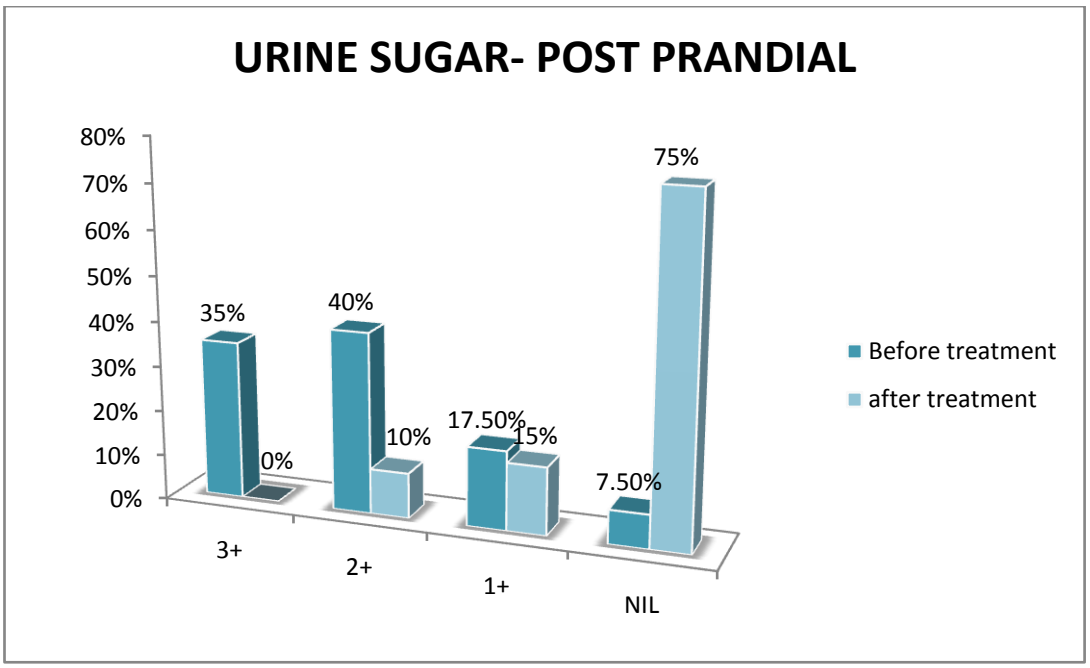


Inference:

From the above chart it may be observed that the Urine sugar position on fasting after treatment had improved drastically,and it was nil in 82.5% of the cases after treatment.

(B)URINE SUGAR – POST PRANDIAL

URINE SUGAR	BEFORE TREATMENT	PERCENTAGE %	AFTER TREATMENT	PERCENTAGE%
+++	14	35%	0	0%
++	16	40%	4	10%
+	7	17.5%	6	15%
NIL	3	7.5%	30	75%

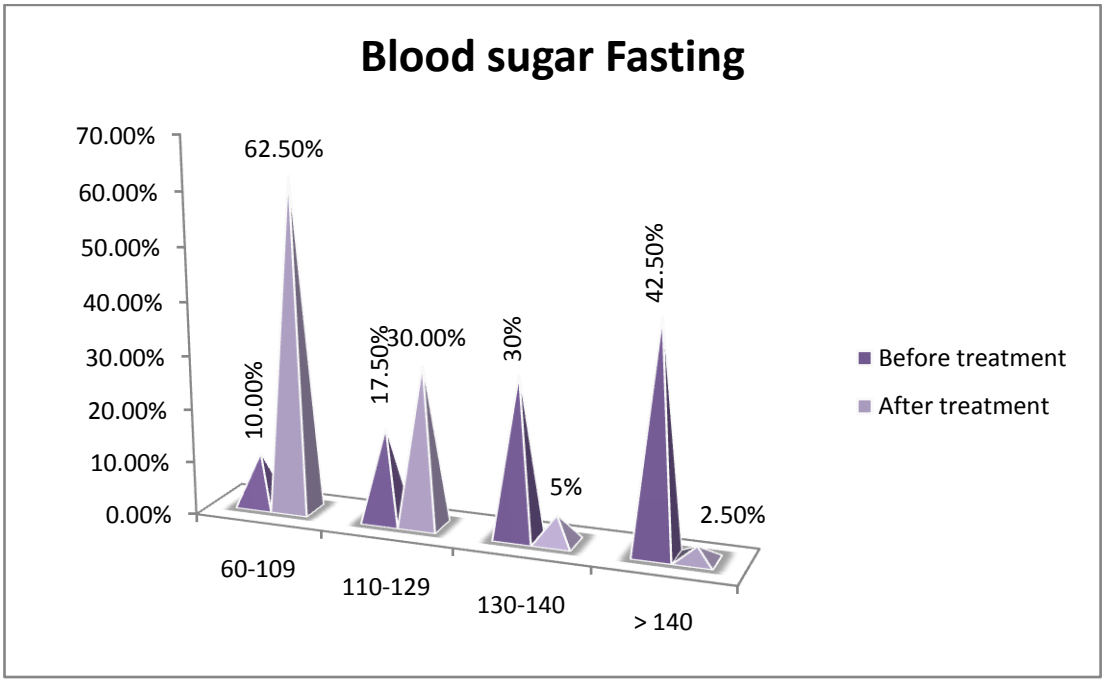


Inference:

It may be noted that the Post prandial urine sugar position after treatment had improved drastically it was nil in 75% of the cases after treatment.

19.(A) BLOOD SUGAR - FASTING

BLOOD SUGAR FASTING (mg)	BEFORE TREATMENT NO.OF CASES	ERCENTAGE %	AFTER REATMENT NO.OF CASES	ERCENTAGE%
60 – 109	4	10%	25	62.5%
110 – 129	7	17.5%	12	30%
130-149	12	30%	2	5%
Above 150	17	42.5%	1	2.5%

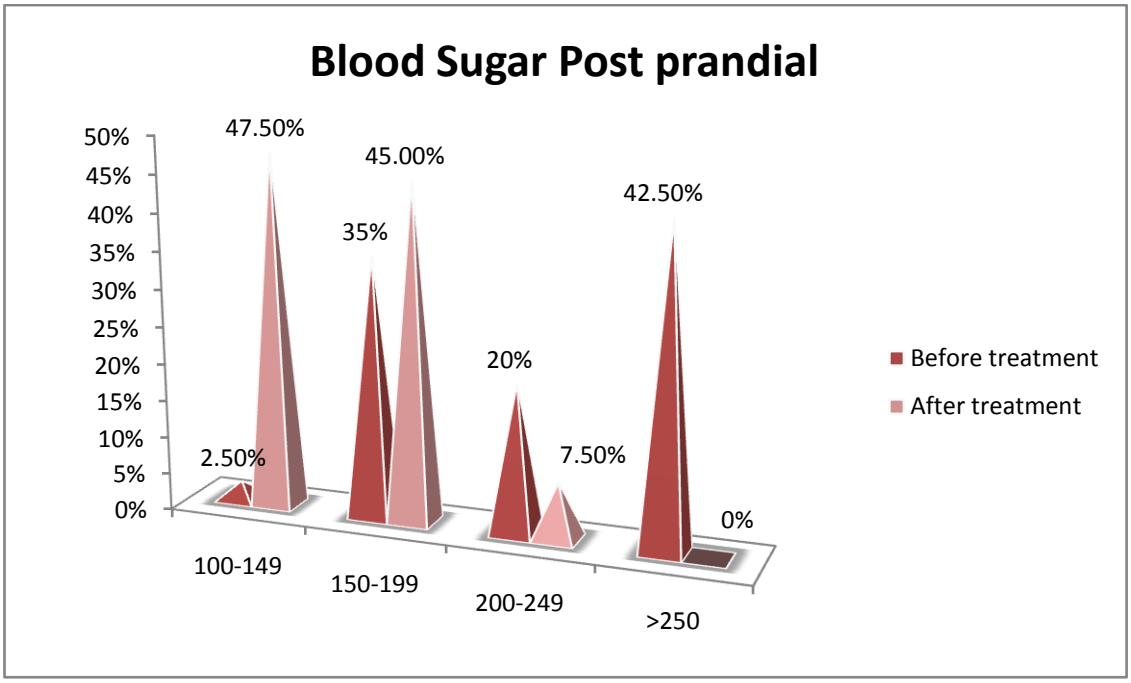


Inference:

Fasting blood sugar has control in 62.5% of the cases.

(B)BLOOD SUGAR POST PRANDIAL

LOOD SUGAR OST PRANDIAL (mg)	BEFORE TREATMENT NO. OF CASES	PERCENTAGE %	AFTER TREATMENT NO.OF CASES	PERCENTAGE%
100 – 149	1	2.5%	19	47.5%
150 - 199	14	35%	18	45%
200 – 249	8	20%	3	7.5%
250 – above	17	42.5%	0	0%

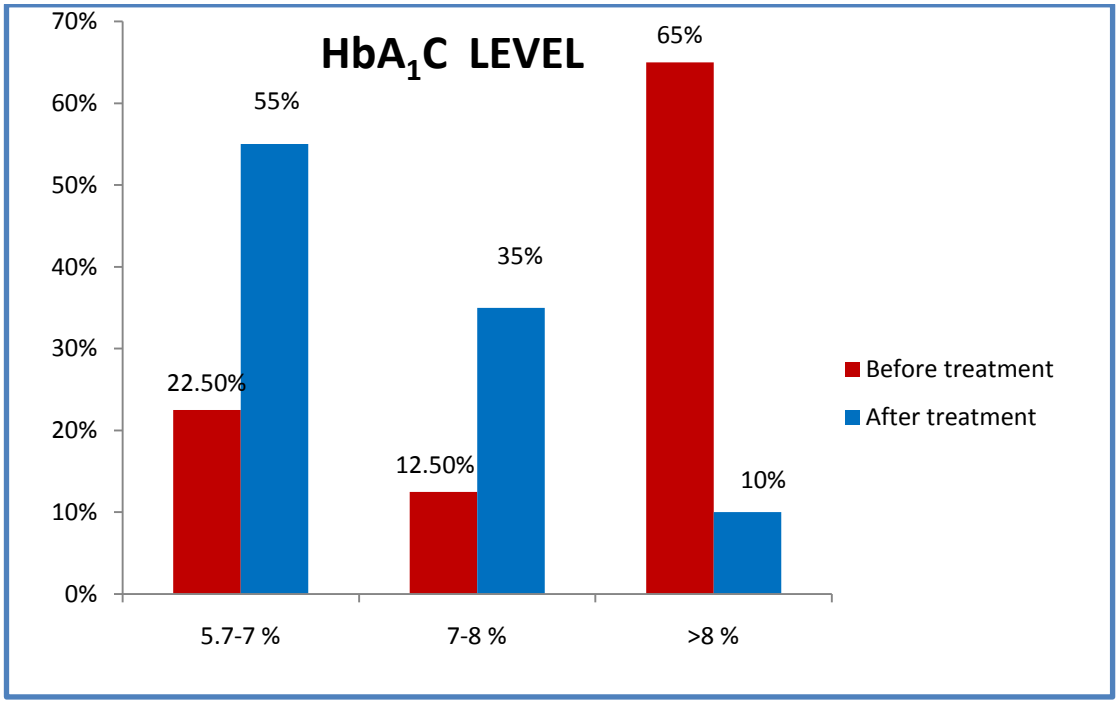


Inference:

The blood sugar Post prandial level has control in 70% of the cases.

20.HBA$_1$C LEVEL

HBA$_1$C	Before treatment no. of cases	Percentage %	After treatment no.of cases	Percentage%
Good control 5.7-6.9%	9	22.5%	22	55%
Fair control 7-8%	5	12.5%	14	35%
Poor control >8.1%	26	65%	4	10%

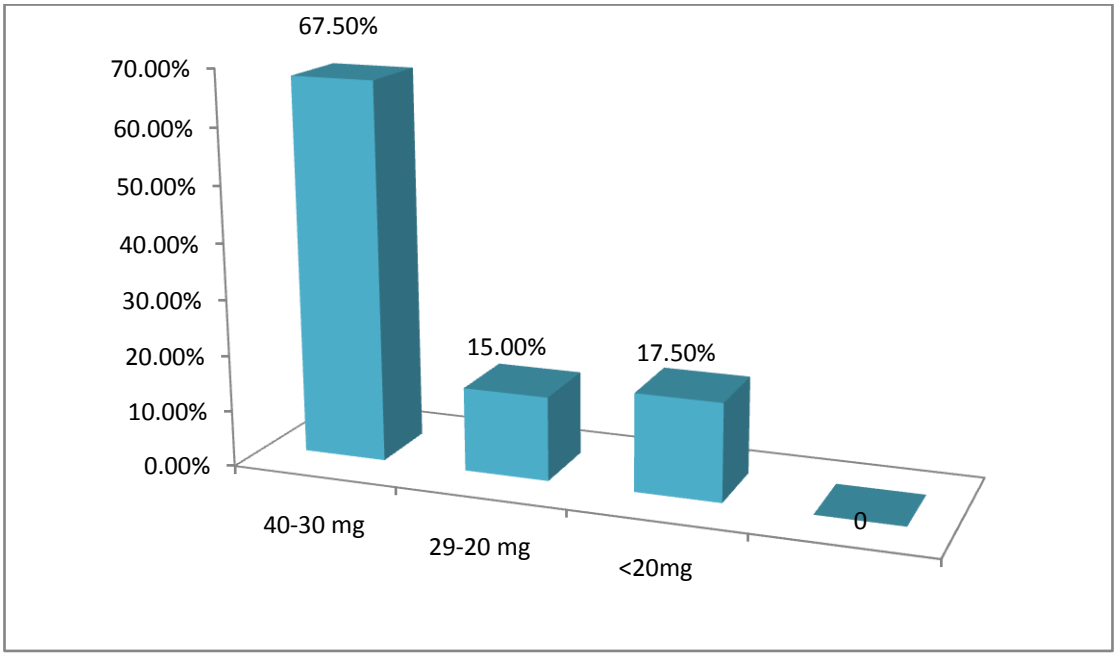


Inference:

HbA$_1$C level has good control in 55% of patients, fair control in 35% of patients and poor control in 10% of patients.

21.(A) BASED ON FASTING

S.no	Blood Sugar level Fasting	Prognosis	No.of Patients	Percentage
1	40-30mg	Good	27	67.5%
2	29-20mg	Moderate	6	15%
3	<20mg	Mild	7	17.5%

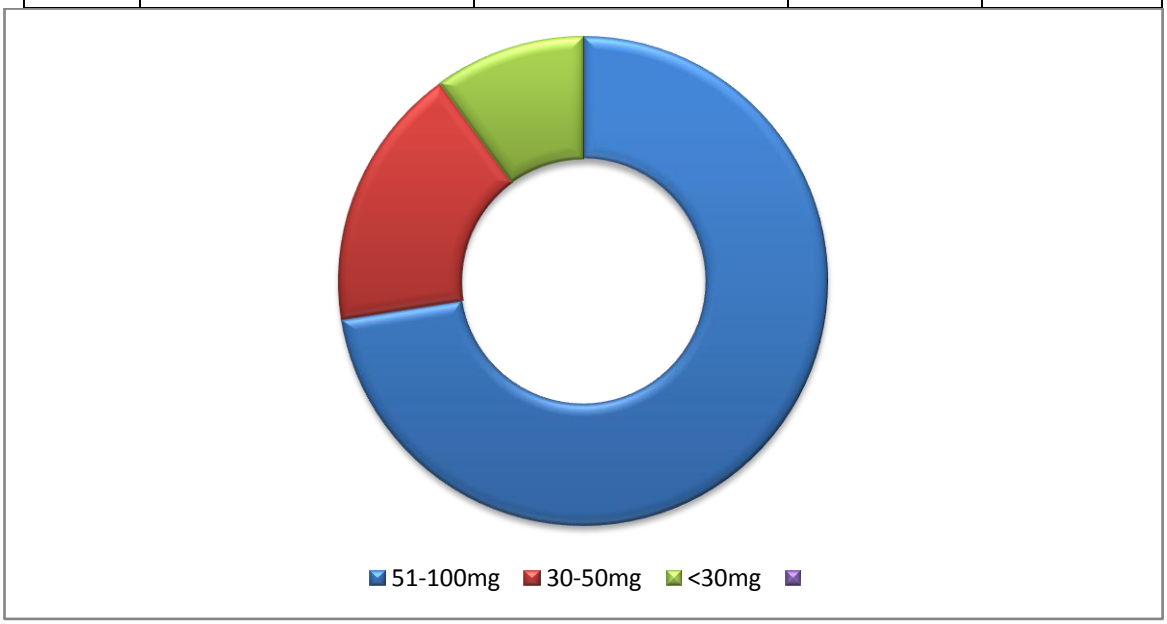


Inference:

Of the 40 patients, 27patients(67.5%) showed good result,6 patients (15%)showed moderate result and 7 patients(17.5%) showed mild result.

(B) BASED ON POST PRANDIAL

S.no	Blood Sugar level Post prandial	Prognosis	o.of Patients	Percentage
1	51-100mg	Good	29	72.5%
2	30-50mg	Moderate	7	17.5%
3	<30mg	Mild	4	10%



BLOOD SUGAR LEVEL

S.No	Name	ge/Sex	Blood Sugar Level			
			Before treatment		**After treatment**	
			F	**PP**	**F**	**PP**
1.	Babu	57/M	166	262	116	240
2	Paramanantham	59/M	95	276	95	180
3	Kuppammal	58/F	162	258	114	182
4	Elumalai	42/M	139	299	96	140
5	Selva kumar	54/M	136	280	105	140
6	Kasturi	67/F	127	172	95	130
7	Renuga devi	42/F	163	298	109	138
8	Mohanam	55/M	168	242	110	160
9	Mabubasha	52/M	199	289	180	249
10	Anandhi	44/F	139	199	90	142
11	jagadesh	36/M	151	187	110	140
12	Subathra	50/F	158	256	110	176
13	Regina mary	58/F	190	272	140	191
14	suresh	49/M	136	190	96	126
15	Mahalakshmi	41/F	136	190	98	142
16	Manjula	40/F	152	241	102	162
17	Rajendran	39/M	189	289	100	180
18	Malliga	50/F	109	142	80	120
19	Sasikala	45/F	104	213	90	150
20	Kannan	37/M	126	212	94	138
21	Sujatha	46/F	181	288	98	142
22	Shanmugam	53/M	96	168	80	140
23	Sibu	45/M	117	170	100	120
24	Ganesan	43/M	120	180	105	150
25	Shanmugapriya	36/F	157	263	110	160
26	Babu	48/M	110	160	100	130
27	Harikrishnan	42/M	139	181	109	130
28	Siva	46/M	168	299	140	180
29	Angamuthu	44/M	140	256	110	160
30	Gowri shankar	39/M	120	168	90	142
31	Saroja	55/F	133	249	100	160
32	Kumar	53/M	162	258	120	160
33	Vasantha	46/F	198	311	120	200
34	Chandran	49/M	131	169	97	131
35	Kalalakshmi	52/F	140	210	100	160
36	Rajagopal	56/M	170	200	120	150
37	Prabakaran	47/M	158	195	108	160
38	Thilagavathi	58/F	142	194	110	140
39	Selvi	60/F	128	278	84	132
40	Yuvaprasad	53/M	130	210	110	160

BMI CHART OF THE PATIENTS

Sl.no	Name	Age/Sex	Weight (kg)	Height (m)	BMI	ormal/Lean/Overweight
1	Babu	57/M	62	1.62	22	N
2	Paramanantham	59/M	58	1.60	23	N
3	Kuppammal	58/F	60	1.63	23	N
4	Elumalai	42/M	70	1.65	26	O
5	Selva kumar	54/M	59	1.58	23	N
6	Kasturi	67/F	59	1.63	22	N
7	Renuga devi	42/F	68	1.73	22	N
8	Mohanam	55/M	62	1.60	24	N
9	Mabubasha	52/M	65	1.70	22	N
10	Anandhi	44/F	63	1.63	24	N
11	jagadesh	36/M	50	1.64	18	L
12	Subathra	50/F	80	1.75	26	O
13	Regina mary	58/F	70	1.63	26	O
14	suresh	49/M	70	1.89	24	N
15	Mahalakshmi	41/F	65	1.63	24	N
16	Manjula	40/F	60	1.62	22	N
17	Rajendran	39/M	70	1.70	24	N
18	Malliga	50/F	75	1.70	26	O
19	Sasikala	45/F	56	1.57	22	N
20	Kannan	37/M	61	1.65	22	N
21	Sujatha	46/F	63	1.63	24	N
22	Shanmugam	53/M	55	1.55	22	N
23	Sibu	45/M	70	1.70	24	N
24	Ganesan	43/M	50	1.63	18	L
25	Shanmugapriya	36/F	56	1.57	22	N
26	Babu	48/M	61	1.65	22	N
27	Harikrishnan	42/M	58	1.63	22	N
28	Siva	46/M	75	1.70	27	O
29	Angamuthu	44/M	50	1.63	18	L
30	Gowri shankar	39/M	61	1.65	21	N
31	Saroja	55/F	59	1.63	22	N
32	Kumar	53/M	55	1.55	22	N
33	Vasantha	46/F	72	1.70	24	N
34	Chandran	49/M	50	1.63	18	L
35	Kalalakshmi	52/F	61	1.68	21	N
36	Rajagopal	56/M	50	1.63	18	L
37	Prabakaran	47/M	70	1.65	26	O
38	Thilagavathi	58/F	62	1.65	24	N
39	Selvi	60/F	61	1.65	21	N
40	Yuvaprasad	53/M	59	1.63	22	N

BIOCHEMICAL ANALYSIS REPORT OF THE PATIENTS

SN NO	O.P NO	Name	Age / Sex	Blood sugar (mg)				Urine sugar				HbA1C	
				BT		AT		BT		AT		BT	AT
				F	PP	F	PP	F	PP	F	PP		
1	4846	Babu	57/M	166	262	116	240	++	+++	+	++	8.9	7.8
2	7150	Paramanantham	59/M	95	276	95	180	NIL	+++	NIL	+	11.2	7.2
3	3479	Kuppammal	58/F	162	258	114	32	+	++	+	+	8.3	6.9
4	435	Elumalai	42/M	139	299	96	140	+	+++	NIL	NIL	9.6	6.2
5	3234	Selva kumar	54/M	136	280	105	140	+	+++	NIL	NIL	9.5	6.4
6	9440	Kasturi	67/F	127	172	95	130	NIL	NIL	NIL	NIL	6	5.8
7	6020	Renuga devi	42/F	163	298	109	138	++	+++	NIL	NIL	10.2	8.2
8	7157	Mohanam	55/M	168	242	110	160	++	++	NIL	+	10.4	8.1
9	2010	Mabubasha	52/M	199	289	180	249	++	+++	+	++	11.6	8.8
10	8963	Anandhi	44/F	139	199	90	142	+	++	NIL	NIL	8	6.5
11	3583	jagadesh	36/M	151	187	110	140	+	++	NIL	NIL	7.7	6.2
12	402	Subathra	50/F	158	256	110	176	+	++	NIL	+	9.1	7.6
13	2536	Regina mary	58/F	190	272	140	191	++	+++	+	++	11.2	7.8
14	2443	suresh	49/M	136	190	96	126	+	++	NIL	NIL	8.3	6.9
15	7432	Mahalakshmi	41/F	136	190	98	142	+	++	NIL	NIL	6.9	6.1
16	3565	Manjula	40/F	152	241	102	162	+	++	NIL	NIL	9.2	7.1
17	5471	Rajendran	39/M	189	289	100	180	++	+++	NIL	+	11	7.3
18	7899	Malliga	50/F	109	142	80	120	NIL	+	NIL	NIL	6.5	6.1
19	5045	Sasikala	45/F	104	213	90	150	NIL	++	NIL	NIL	6.69	6.20
20	3169	Kannan	37/M	126	212	94	138	NIL	++	NIL	NIL	9.5	4.9

Sl.No	).P NO	Name	ge / Sex	Blood sugar (mg)				Urine sugar				HbA1C	
				BT		AT		BT		AT		BT	AT
				F	PP	F	PP	F	PP	F	PP		
21	3368	Sujatha	46/F	181	288	98	142	++	+++	NIL	NIL	9.8	7.1
22	49	Shanmugam	53/M	96	168	80	140	NIL	+	NIL	NIL	6.4	6.1
23	8738	Sibu	45/M	117	170	100	120	NIL	NIL	NIL	NIL	6.7	6.2
24	9398	Ganesan	43/M	120	180	105	150	NIL	+	NIL	NIL	6.5	6.1
25	9946	Shanmugapriya	36/F	157	263	110	160	+	+++	NIL	NIL	8.21	6.2
26	307	Babu	48/M	110	160	100	130	NIL	NIL	NIL	NIL	6.7	6.1
27	4905	Harikrishnan	42/M	139	181	109	130	+	+	NIL	NIL	8.1	6.1
28	6643	Siva	46/M	168	299	140	180	++	+++	+	+	13.1	8.5
29	3360	Angamuthu	44/M	140	256	110	160	+	+++	NIL	NIL	8.9	6.5
30	1177	Gowri shankar	39/M	120	168	90	142	NIL	+	NIL	NIL	6.8	6.1
31	899	Saroja	55/F	133	249	100	160	+	++	NIL	NIL	7.6	6.4
32	3376	Kumar	53/M	162	258	120	160	+	++	+	NIL	10.1	7.2
33	1096	Vasantha	46/F	198	311	120	200	++	+++	+	++	10.3	7.2
34	2130	Chandran	49/M	131	169	97	131	NIL	+	NIL	NIL	7.2	6.2
35	5047	Kalalakshmi	52/F	140	210	100	160	+	++	NIL	NIL	8.3	7.1
36	8762	Rajagopal	56/M	170	200	120	150	++	++	NIL	NIL	8.1	7
37	3311	Prabakaran	47/M	158	195	108	160	+	++	NIL	NIL	8.1	6.7
38	9666	Thilagavathi	58/F	142	194	110	140	+	+	NIL	NIL	8.3	7
39	)665	Selvi	60/F	128	278	84	132	NIL	+++	NIL	NIL	9.1	6.8
40	3183	Yuvaprasad	53/M	130	210	110	160	+	++	NIL	NIL	7.9	6.5

BT – Before Treatment, AT – After Treatment, N – Nil F- Fasting, PP- Post Prandial,

LABORATORY INVESTIGATION REPORT OF THE PATIENTS

S.no	O.P no	Name	Age / Sex	Before treatment				After Treatment				ESR (mm)				Hb (gms%		Urine Anal		
				TC	DC			TC	DC			BT		AT		BT	AT	BT		
				(cu/mm	P%	L%	E%	cu/mm	P%	L%	E%	½ hr	1 hr	½ hr	1 hr			Alb	Dep	Alb
1	4846	Babu	57/M	9400	57	39	4	9500	58	39	3	3	18	3	10	10	10.5	N	FEC	N
2	7150	Paramanantham	59/M	9500	58	38	4	9600	60	37	3	6	10	5	6	11	12	N	FEC	N
3	3479	Kuppammal	58/F	9200	56	42	2	9400	62	36	2	8	10	4	5	11.6	12	N	FPC	N
4	435	Elumalai	42/M	9800	62	35	3	9700	63	36	1	8	12	4	10	13	13.8	N	FEC	N
5	3234	Selva kumar	54/M	9600	62	33	5	9700	60	35	5	3	5	5	7	8	9.9	N	FPC	N
6	9440	Kasturi	67/F	9700	59	37	4	9800	60	38	2	12	20	7	15	10	10.6	N	FEC	N
7	6020	Renuga devi	42/F	9800	58	37	5	9900	61	36	3	5	10	3	8	11	11.6	N	FEC	N
8	7157	Mohanam	55/M	9400	66	29	5	9500	67	32	1	10	20	5	10	10	11	N	FPC	N
9	2010	Mabubasha	52/M	8400	56	36	8	8600	58	38	4	2	8	2	4	9	10	N	FEC	N
10	8963	Anandhi	44/F	9100	62	31	7	8900	61	37	2	3	10	5	10	10	11	N	FEC	N
11	3583	jagadesh	36/M	8600	54	44	2	8700	56	42	2	12	22	10	6	10	10.9	N	FPC	N
12	402	Subathra	50/F	8200	59	34	7	8300	59	40	1	7	12	3	10	11	12	N	FEC	N
13	2536	Regina mary	58/F	9900	66	32	2	9800	61	37	2	3	5	3	10	12	12.6	N	FPC	N
14	2443	suresh	49/M	8400	63	37	3	8600	58	39	3	5	10	5	8	12	12.5	N	FEC	N
15	7432	Mahalakshmi	41/F	9400	62	32	4	9500	63	34	3	4	8	3	8	10	10.4	N	FPC	N
16	3565	Manjula	40/F	8800	56	38	6	8900	61	37	2	10	20	5	10	8.8	9.4	N	FPC	N
17	5471	Rajendran	39/M	9600	63	33	4	9800	64	34	2	7	14	5	10	11	12	N	FEC	N
18	7899	Malliga	50/F	9500	62	36	2	9600	63	35	2	2	10	2	8	12	12.4	N	FEC	N
19	5045	Sasikala	45/F	9700	56	40	4	9600	59	39	2	6	12	3	6	10	10.2	N	FPC	N
20	3169	Kannan	37/M	9400	60	36	4	9500	61	38	1	16	20	8	12	9.8	9.6	N	FEC	N

Sl. no	O.P no	Name	Age / S	Before treatment				After Treatment				ESR (mm)				Hb (gms%		Urine Ana		
				TC	DC			TC	C			BT		AT			AT	BT		
				(cu/mn	P%	L%	E%	(cu/mn	P%	L%	E%	½ hr	1 hr	½ hr	1 hr	BT		Alb	Dep	Al
21	3368	Sujatha	46/F	10100	60	32	8	10000	65	32	3	12	24	6	12	12	12.6	N	FEC	N
22	49	Shanmugam	53/M	9600	61	37	2	9700	62	36	2	8	14	4	10	10	10.6	N	FPC	N
23	8738	Sibu	45/M	9000	55	39	6	9200	59	39	2	2	8	2	6	11	11.2	N	FEC	N
24	9398	Ganesan	43/M	9200	56	42	2	9300	58	40	2	3	8	3	6	12	12.4	N	FEC	N
25	9946	Shanmugapriya	36/F	8100	61	37	2	8200	61	38	1	4	10	6	8	9	10	N	FEC	N
26	307	Babu	48/M	8400	55	38	7	8600	60	38	2	8	10	4	8	9	9.8	N	FEC	N
27	4905	Harikrishnan	42/M	9200	56	40	4	9400	58	40	2	12	16	6	8	8.8	9.6	N	FPC	N
28	6643	Siva	46/M	10000	59	35	7	10100	62	35	3	8	12	3	10	12	13	N	FEC	N
29	3360	Angamuthu	44/M	8900	58	40	2	8800	58	41	1	5	10	5	6	14	14.2	N	FEC	N
30	1177	Gowri shankar	39/M	9100	63	31	6	9300	66	32	2	10	16	5	10	12	12.4	N	FEC	N
31	899	Saroja	55/F	9500	62	36	2	9600	61	38	1	3	5	3	6	13	13.4	N	FEC	N
32	3376	Kumar	53/M	8600	62	37	1	8500	61	38	1	4	10	2	6	9	9.8	N	FPC	N
33	1096	Vasantha	46/F	9100	61	37	2	9300	59	38	3	2	4	4	8	12	12.6	N	FEC	N
34	2130	Chandran	49/M	8900	50	44	6	9000	55	43	3	8	10	6	8	9	10	N	FEC	N
35	5047	Kalalakshmi	52/F	9100	63	35	2	9400	61	37	2	4	7	5	10	11	12	N	FEC	N
36	8762	Rajagopal	56/M	9500	52	41	7	9700	56	41	3	3	8	4	12	10	10.4	N	FPC	N
37	3311	Prabakaran	47/M	10200	60	38	2	10300	61	37	2	8	14	6	12	12	13	N	FEC	N
38	9666	Thilagavathi	58/F	9100	55	39	4	9400	57	40	3	8	14	4	10	10	10.2	N	FEC	N
39	665	Selvi	60/F	8600	58	34	8	8900	62	36	2	3	6	3	8	11	12	N	FPC	N
40	3183	Yuvaprasad	53/M	8200	60	36	4	8500	61	36	3	8	16	4	8	10	10.4	N	FPC	N

BT – Before Treatment, AT – After Treatment, N – Nil, TC – Total Blood Count, DC – Differential Blood Count, P – Polymorphs, L – Leucocytes,
E – Eosinophils, ESR – Erythrocytes Sedimentation Rate, mm – Milli meter,Hb – Hemoglobin, Alb – Albumin, Sug – Sugar, Dep – Deposits,
FEC – Few Epithelial cells, FPC – Few Pus cells

DISCUSSION

DISCUSSION

Madhumegam, is a clinical entity described byYugimunivar in his **YugiVaidhyaChintamani 800** can be compared with **Diabetes mellitus**. The classical symptoms are Polyuria,Polyphagia,Polydipsia,Itching all over the body,Pain all over the body ,Emaciation and Peripheral neuritis.

In my study, 40 patients with **Madhumegam** were selected in the Department of PothuMaruthuvam , Government Siddha Medical College, attached to Arignar Anna Govt Hospital for Indian Medicine,Arumbakkam, Chennai -106.

All necessary investigations were carried out to all patients and trial medicine was given. The results of before and after treatment of all the patients were analysed and discussed below.

Age and Kaalam distribution:

It shows that Madhumegam is seen in the age group of 41-50 yrs(45%), 51-60 years (37.5) ,30-40 years(15%)and 2.5% in 61-70 yrs .

Sex distribution:

Out of 40 patients, 23 cases (57.5%) were male and 17 cases (42.5%) were female.

Socio-economic status

Regarding socio-economic status,20 patients (50%) comes under middle class category, 13 patients (32.5%) comes under poor category, and 7 patients (17.5%) comes under high class category.

Occupational status:

Of 40 patients,18 patients(45%) were office going, 7 patients (17.5%) were housewife, 7 patients (17.5%) were labourers and 8 patients (20%) were doing business.

Diet:

32 patients (80%) takes mixed diet and 8 patients (20%) take vegetarian diet.

Body built:

Regarding bodybuilt, 29 patients(72.5%) were having normal weight, 6 patients(15%) were overweight and 5 patients(12.5%) were lean.

Family history:

Regarding Family history, 28 patients(70%) had no family history of Diabetes,6 patient's father(15%) and 6 patient's mother(15%) is diabetic.

Thinai reference:

Out of 40 patients, 36 patients (90%) comes under Neithalnilam and 4 patients(10%) comes under Maruthanilam.

Paruvakaalam:

From selected 40 patients, 21 patient (52.5%) comes under Munpani 2 patients (5%) comes under Koothirkaalam, 6 Patients (15%) comes under Pinpani 3 patients (7.5%) comes under Kaarkaalam, 4 patients comes under Elavenil and Mudhuvenil each. The seasonal variation has no impact on Madhumegam.

Duration of illness:

Out of 40 patients, 16 patients (40%) belongs to 3 to 6 months category,10 patients (25%) belongs to newly identified category, 7 patients (17.5%) belongs to 6 months to one year category and 7 patients (17.5%) belongs to more than one year category.

Mukkutram classification:

Vali:

1. Abanan affected in all patients (100%) causing Polyuria, Nocturia,Constipation.
2. Viyanan affected in all patients (100%)with Pain all over the body.
3. Samanan and Kirukaran affected in all patients(100%) causing Polyphagia.
4. Devethathan affected in all patients(100%) causing disturbed sleep,fatigue.
5. Koorman affected in 6 patients(15%) causing dimness of vision.

Azhal:

1. Analagam affected in all patients(100%) causing polyphagia.
2. Sathagam affected in all patients(100%) causing lassitude.
3. Ranjagam,Aalosagam and Prasagam affected in (20%),(15%),(10%) patients causing pallor,dimness of vision and dry skin respectively.

Iyyam:

1. Kilethagam affected in all patients (100%) results in Polyphagia.
2. Santhigam affected in 26 patients(65%) causing joint pain.

Ezhuudalthathukkal:

1. Saaram affected in all patients results in tiredness,general debility.
2. Senneer affected in all cases causing pallor,dryness.
3. Oon and Kozhuppu affected in 4 patients (10%) each causing emaciation
4. Enbu affected in 28 patients (70%) causing joint pain.

Envagaithervugal:

1. NaaNaadi,andMoothiram affected in all 40 patients(100%).
2. Malam affected in 8 patients (20%)results in constipation.
3. Vizhi was affected in 6 patients dimness of vision,(15%),Sparisam affected in 6 patients(15%) causing dry skin.

Naadi:

26 patients(70%) had PithaVathaNaadi and 8 patients(20%) had VathaPithanaadi and 6 patients(15%) had PithaKabanaadi.

Neikuri:

10 patients (25%) had Kabha neer,20 patients (50%) had Pithaneer, 10 patients (25%) had Vathaneer.

Signs and symptoms:

Polyuria, Polyphagia, Polydipsia, dryness of the mouth and throat and disturbed sleep present in all cases i.e 100%. Pain all over the body in 34 patients (85%), Itching 10

patients (25%), Constipation 8 patients (20%), emaciation 4 patients (10%) and Peripheral neuritis 10 patients (25%). Pruritis vulvae 4 patients (10%).

Clinical prognosis:

The clinical signs and symptoms were improved after treatment showing only 17.5% had polyurea, 12.5% had polyphagia, 10% of the people had polydipsia, 17.5% have pain all over the body, 10% have peripheral neuritis and 15% had disturbed sleep. Pruritis vulvae, itching all over the body, dryness of mouth and constipation were completely relieved.

Laboratory assessment:

Urine sugar Fasting and Postprandial became normal in 82.5% and 75% of patients respectively.

Blood sugar Fasting and Postprandial controlled in 62.5% and 47.5% of patients respectively.

HbA_1C level reduced in 55% of cases which shows good control in Madhumegam.

Investigations:

Investigations like TC, DC, ESR, Hb, Serum cholesterol and Blood urea, were examined and urine analysis for albumin, sugar and deposits were also examined. Blood sugar Fasting,Post prandial, HbA_1C were examined.

Trial medicine:

Naval kottaichoornam 1g,BD with warm water after food was administered for 90 days.

Suvaimukkutram theory:

Madhumegam is due to derangement of Pitham and Kabakutram.The trial drug chosen has Kaippu and thuvarpusuvai which settles down deranged kutrams.

Biochemical analysis:

It reveals the presence of Ferrous iron, calcium, Potassium and alkaloids.

Toxicological study:

Acute oral toxicity study followed as per OECD 423 guidelines and Sub acute oral toxicity study done as per OECD 407 guidelines revealed no toxicity in the trial medicine.

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Pharmacological study:

Pharmacological activity is screened against Streptozotocin induced Diabetes in rat model. This proves that the trial medicine has Anti-diabetic activity.

Clinical Study:

All the patients were treated with **Naval kottaichooranam** for an average of 90days. All necessary laboratory investigations were again repeated, after the completion of treatment and showed significant improvement in clinical manifestations of Madhumegam.

Bio statistical analysis:

The p value is highly significant ($p<0.001$). So there is significant reducing of Fasting and Postprandial blood sugar level (mg) among the patients for the treatment of **Madhumegam.**Hence it is concluded that the treatment was **effective and significant.**

Grading of results:

Out of 40 patients, 70% of cases showed good result, 20% of the cases showed Moderate result and 10% showed mild result.

SUMMARY

SUMMARY

The clinical study on **Madhumegam** was carried out in the Post Graduate department of Maruthuvam,Govt Siddha Medical College,Arignar Anna Hospital, Chennai-106, during the period of 2014-2016.

A total of 40 patients were treated in the O.P and I.P department. the clinical and pathological assessment was carried out on the basis of both Siddha and Modern aspects.

All the 40 patients were treated with **naval kottai Chooranam**, 1g BD with warm water for 90 days. The responses were assessed once in 15 days for all the patients.

- The peak incidence of Madhumegam was in 51-60 yrs (37.5%) in both sexes.
- The prevalence was higher among the middle class 50%, poor 32.5% and high class population 17.5%.
- Out of 40 patients, 45% were office going, housewives 17.5%,, labourers17.5% and business people were 20%.
- Regarding diet, 80% takes mixed diet and 20% takes vegetarian diet.
- Regarding family history 70% had no relavent history,15% of father is diabetic and 15% of mother is diabetic.
- Out of 40 patients,92.5% comes underPitha kaalam,5% comes under Vatha kaalam and 2.5% comes under Kaba kaalam
- In Vatham-Abanan,Viyanan,Samanan,Kirukaran and Devathathan were affected 100%,Koorman 15% was affected.
- In Pitham-Analagam and Sathagam were 100% affected.Ranjagam(20%),Alosagam (15%),prasagam(10%) were affected.
- In Kabam, Kilethagam(100%) and Santhigam(65%) were affected.
- Among the EzhuudalthathukkalSaaram,Seneer were 100% affected,Enbu(70%), Oon(10%),Kozhuppu(10%) were affected.

- Regarding EnvagaithervugalNaaNaadi, and Moothiram were 100% affected Malam 20%,Vizhi(15%) Sparisam(15%) were affected.
- Naadi in Madhumegam was PithaVathanaadi(65%), VathaPithanaadi 20% and PithaKabam 15%.
- In Neikuri examination 25% showed Vathaneer, 50% had pithaneer and 25% showed kabaneer.
- The clinical trial shows that there is significant improvement in clinical manifestations of Madhumegam.
- Urine sugar Fasting and Postprandial became normal in 82.5% and 75% of patients respectively.
- Regarding Blood sugar level fasting and post prandial blood sugar reduced in 62.5% and 47.5% of the cases.
- HbA_1C level improved in 55% of cases (5.7-7%) which shows good control in **Madhumegam**.
- The Ingredients of trial medicine have the properties to control Blood sugar level.
- The Toxicological study of trial medicine revealed no toxicity.
- The Pharmacological study shows anti diabetic activity in Streptozotocin induced diabetic rats.
- The Bio-statistical report of the clinical trial shows significant p value< 0.001 and hence the treatment was effective and significant.
- Among 40 cases, 67.5% of the patients showed good result, 15% moderate result and 17.5% showed mild result.

CONCLUSION

CONCLUSION

- **Madhumegam**is primarily due to derangement of Pitham and Kabam
- The trial medicine **naval kottaichooranam** has property of neutralizing deranged Kutrams.
- From preclinical studies the trial medicine revealed no toxicity in animal model and proved to be safe in human subjects.
- From preclinical Pharmacological studies, the trial medicine has anti-diabetic activity.
- During the clinical study no contraindication was reported
- The trial medicine significantly reduced blood sugar level and other symptoms.
- The trial medicine is cost effective and palatable.

Hence I conclude that trial drug will be a better drug that can be used in the treatment of **Madhumegam.**

ANNEXURE

CERTIFICATES

The Tamil Nadu Dr.M.G.R. Medical University

#69, Anna salai, Guindy, Chennai-600 032.

This certificate is awarded to

Dr./~~Mr.~~/~~Ms.~~ LATHA RANI. M

for participating as Resource Person / Delegate in the Fourteenth Workshop on

"Research Methodology & Biostatistics"

for AYUSH Post Graduates & Researchers

Organised by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University from 5th to 9th May 2014.

Dr. N. KABILAN M.D. (Siddha)
Reader, Dept. of Siddha

Dr. JHANSI CHARLES, M.D.
Registrar

Prof. Dr. D. SHANTHARAM, M.D., D.Diab.,
Vice-Chancellor

GOVERNMENT SIDDHA MEDICAL COLLEGE
Arumbakkam, Chennai-106

Communication Of The Decision Of Institutional Ethical Committee (IEC)

IEC No: GSMC-CH-ME-3/003/2014

Protocol title: AN OPEN CLINICAL STUDY ON MADHUMEGAM (DIABETES MELLITUS) WITH THE EVALUATION OF SIDDHA DRUG NAVAL KOOTAI CHOORANAM	
Principal Investigator:	DR. M. LATHA RANI
Name & Address of Institution: Government Siddha Medical College, Arumbakkam, Chennai-106	
☐ New Review	☐ Revised Review ☐ Expedited Review
Date of review (DD/MM/YY):	13-06-2014
Date of Previous Review, If Revised Application:	
Decision of the IEC	
☑ Recommended	☐ Recommended with suggestions
☐ Revision	☐ Rejected
Suggestions / Reasons / Remarks: (i) In clinical assesment, GTT should be excluded (ii) Blood Sugar fasting, PP and HbA1c should be included in primary outcome.	
Recommended for a period of 1 year from date of completion of preclinical studies :	

Please Note:

- Inform IEC immediately in case of any adverse events/serious drug reaction.
- Seek IECapproval in case of any change in the study procedure, site and investigator
- This approval is valid only for period mentioned above
- IEC member have the right to review the trial with prior intimation.

Dr.P.Jeyaprakashnarayanan
Chairman

Dr.V.Banumathi
Member Secretary

INSTITUTIONAL ETHICAL COMMITTEE

Date:

Sub: IEC review of research proposals.

Ref: Your letter dated

MEMBERS	PARTICIPATION	SIGNATURE
DR.P.JEYAPRAKASH NARAYANAN M.D(S)., Chairman	☐	
DR.V.BANUMATHI M.D(S)., Member Secretary	☐	
DR.N.KABILAN M.D(S)., Clinician- Siddha	☑	
DR.P.SATHIYA RAJESWARAN M.D(S)., Clinician- Siddha	☑	
DR.G.AADINATH REDDY,M.Pharm, Ph.D., Pharmacologist	☑	
DR.S.THILAGAVATHY Msc.,Ph.D., Social Scientist	☑	
DR.T.MAHALAKSHMI M.A.,Ph.D., Linguistic Expert	☑	
DR.P.VIDYA M.B.B.S., DMRD., Modern Medicine Expert	☑	
MR.P.SARAVANAN., Public Person	☑	

13/6/14.

Dr.P.Jeyaprakashnarayanan M.D(S).,
Chairman

Dr.V.Banumathi M.D(S).,
Member Secretary

Certificate

This is certify that the project titled Toxicological and pharmacological activity of NAVALKOTTAI CHOORANAM in ratshas been approved by the

IAEC No: IAEC/XLIV/19/CLBMCP/2014

Name of Chairman/ Member Secretary IAEC:

08/05/14

Signature with date

Dr. Baid Mehta College of Pharmacy CHENNAI-97

C.L.BAID METHA COLLEGE OF PHARMACY

(An ISO 9001-2000 certified institute)

Jyothi Nagar, Old Mahabalipuram Road

Thoraipakkam, Chennai – 600 097

CERTIFICATE

This is to certify that the project entitled, **Toxicological and Pharmacological study on NAVALKOTTAI CHOORANAM** in rats submitted in partial fulfilment for the degree of **M.D. (siddha)** was carried out at C.L. Baid Metha college of Pharmacy, Chennai-97, in the Department of Pharmacology during the academic year of 2014-2015

(Dr.P.Muralidharan)

Mr. P.Muralidharan, M.Pharm, Ph.D
Professor and Head
Department of Pharmacology,
C.L.Baid Metha college of pharmacy,
Chennai-97

Baid Metha College of Pharmacy Chennai-97

சித்த மருத்துவ மைய ஆராய்ச்சி நிலையம், அரும்பாக்கம், சென்னை - 600 106
सिध्द केन्द्रीय अनुसंधान संस्थान, अरुम्बाक्कम, चेन्नई- 600 106
Siddha Central Research Institute
Arignar Anna Govt. Hospital Campus, Arumbakkam, Chennai-600 106
(Central Council for Research in Siddha, Department of AYUSH,
Ministry of Health & Family Welfare, Govt. of India)
Phone: 044-26214925, Tele Fax: 044-26214809, E.mail: crisiddha@gmail.com, Web: www.crisiddha.tn.nic.in

22nd February 2016

CERTIFICATE

Certified that the drugs submitted for identification by Dr. M. Latha Rani, PG III year, Department of Pothu maruthuvam, Government Siddha Medical College, Chennai- 600 106, are identified as

1. Katukkai thol - *Terminalia chebula* Retz. (Pericarp)
2. Nelli vatral - *Phyllanthus emblica* L. (Dried fruit)
3. Aavarai poo - *Senna auriculata* (L.) Roxb. (Flower)
   Syn. *Cassia auriculata* L.
4. Konrai poo - *Cassia fistula* L. (Flower)
5. Sirukurinjan ilai - *Gymnema sylvestre* (Retz.) R. Br.ex.Sm. (Leaf)
6. Naval kottai - *Syzygium cumini* (L.) Skeels (Seed)
7. Manjal - *Curcuma longa* L. (Rhizome)
8. Koraik kilanku - *Cyperus rotundus* L. (Rhizome)

Sasikala Ethirajulu
Consultant (Pharmacognosy)

22/2/16
P.Sathiyarajeswaran
Assistant Director Incharge

PHYSICO CHEMICAL ANALYSIS

சித்த மருத்துவ மைய அராய்ச்சி நிலையம், சென்னை — 600 106
सिद्ध केंद्रीय अनुसन्धान संस्थान, अण्णा सरकारी अस्पताल परिसर, अरुम्बाक्कम, चेन्नई - 600106

SIDDHA CENTRAL RESEACH INSTITUTE

(Central Council for Research in Siddha, Ministry of AYUSH, Govt. of India)
Anna Govt. Hospital Campus, Arumbakkam, Chennai – 600106
Phone: 044-2621 4925, Fax: 044-2621 4809
www.crisiddha.tn.nic.in, Email: crisiddha@gmail.com

06.06.2016

Name of the student: by Dr. M. Latha Rani, III year MD Student,

Department of Pothu Maruthuvam, Government Siddha Medical College, Chennai-600 106

PHYSICO-CHEMICAL ANALYSIS OF NAVALKOTTAI CHOORANAM

S.No	Physicochemical Parameter		Mean
1.	Loss on Drying at 105°C	:	8.8 %
2.	Total Ash	:	5.95 %
3.	Water soluble Ash	:	2.82 %
4.	Acid insoluble Ash	:	1.02 %
5.	Water Soluble Extractive	:	12.7 %
6.	Alcohol Soluble Extractive	:	13.5 %
7.	pH	:	4.2
TLC/HPTLC		:	Annexed

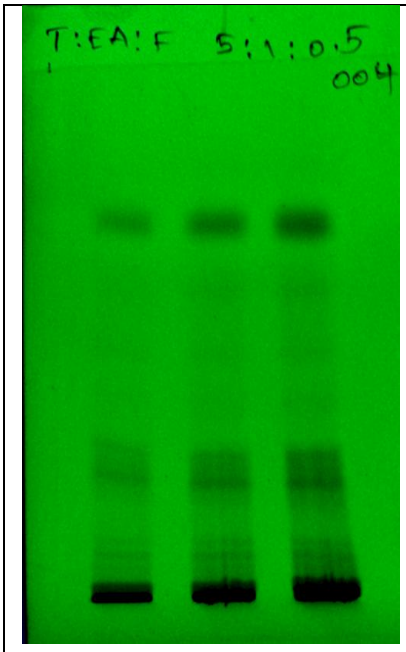
(R. Shakila)
Research Officer (Chemistry)

(Dr. P. Sathiyarajeswaran)
Assistant Director (Scientist 2) I/c

Dr. P. SATHIYARAJESWARAN
Assistant Director (Scientist-2) 1/C
Siddha Central Research Institute (CCRS)
Min. of AYUSH, Govt. Of India
Arumbakkam, Chennai-600 106.

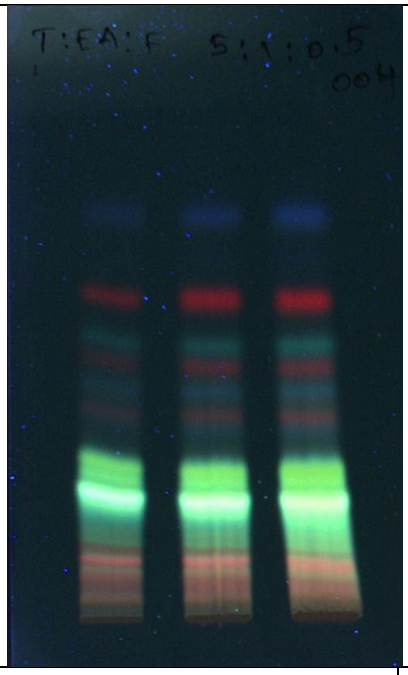
HPTLC REPORT OF NAVALKOTTAI CHOORANAM

Solvent system : Toluene : Ethyl acetate : Formic acid (5:1:0.5)



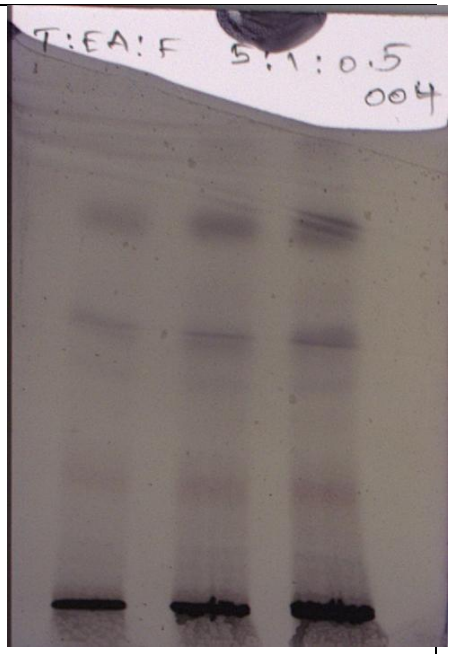
UV 254 nm

R_f	Color
0.08	Dark
0.11	Dark
0.22	Dark
0.27	Dark
0.69	Dark



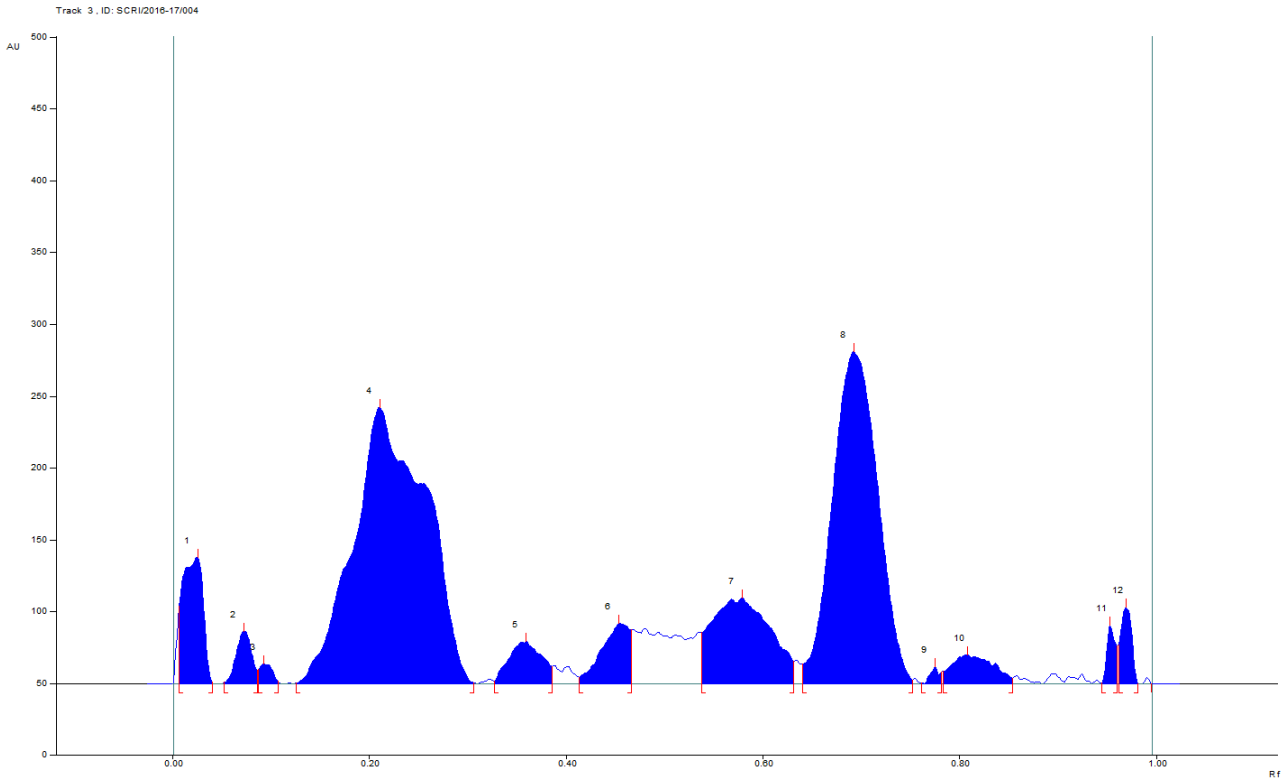
UV 366 nm

R_f	Color
0.04	Brown
0.06	Light Pink
0.1	Pink
0.21	Florescent Green
0.26	Green
0.34	Light Green
0.37	Light Red
0.41	Light Green
0.45	Red
0.49	Green
0.57	Red
0.72	Blue



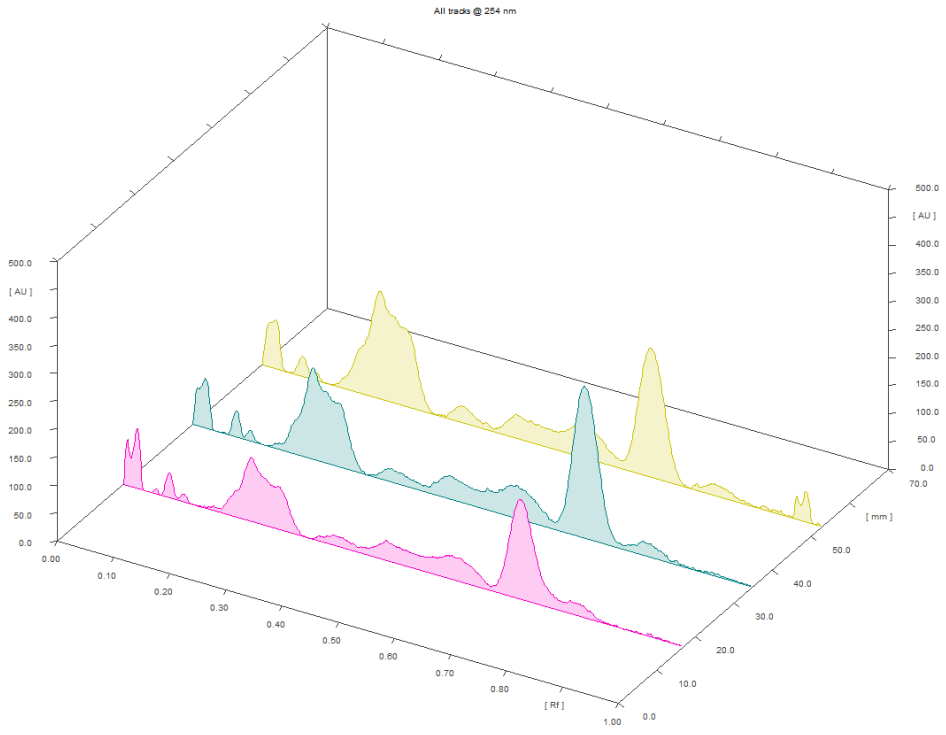
After dipping in Vanillin-sulphuric acid reagent

R_f	Color
0.20	Light Pink
0.40	Light Blue
0.49	Dark
0.69	Dark



Track 3, ID: SCRI/2016-17/004

Peak	Start Position	Start Height	Max Position	Max Height	Max %	End Position	End Height	Area	Area %
1	0.01 Rf	54.0 AU	0.03 Rf	88.2 AU	10.77 %	0.04 Rf	1.6 AU	1729.0 AU	5.27 %
2	0.05 Rf	1.3 AU	0.07 Rf	36.5 AU	4.46 %	0.09 Rf	9.5 AU	538.2 AU	1.64 %
3	0.09 Rf	10.2 AU	0.09 Rf	13.6 AU	1.65 %	0.11 Rf	1.8 AU	170.9 AU	0.52 %
4	0.13 Rf	0.4 AU	0.21 Rf	192.5 AU	23.50 %	0.31 Rf	0.9 AU	13233.6 AU	40.37 %
5	0.33 Rf	2.0 AU	0.36 Rf	29.4 AU	3.58 %	0.39 Rf	12.2 AU	928.5 AU	2.83 %
6	0.41 Rf	4.3 AU	0.45 Rf	42.3 AU	5.16 %	0.47 Rf	37.5 AU	1123.5 AU	3.43 %
7	0.54 Rf	35.7 AU	0.58 Rf	59.6 AU	7.27 %	0.63 Rf	15.5 AU	3333.9 AU	10.17 %
8	0.64 Rf	13.8 AU	0.69 Rf	231.1 AU	28.20 %	0.75 Rf	2.6 AU	9923.0 AU	30.27 %
9	0.76 Rf	0.7 AU	0.78 Rf	11.8 AU	1.44 %	0.78 Rf	8.0 AU	102.3 AU	0.31 %
10	0.78 Rf	8.5 AU	0.81 Rf	20.3 AU	2.48 %	0.85 Rf	4.0 AU	805.9 AU	2.46 %
11	0.95 Rf	0.7 AU	0.95 Rf	40.6 AU	4.95 %	0.96 Rf	26.3 AU	334.2 AU	1.02 %
12	0.96 Rf	27.3 AU	0.97 Rf	53.6 AU	6.54 %	0.98 Rf	0.1 AU	558.6 AU	1.70 %


All tracks @ 254 nm
500.0
[AU]
400.0
350.0
300.0
250.0
200.0
150.0
100.0
50.0
0.0
0.00
0.10
0.20
0.30
0.40
0.50
0.60
0.70
0.80
[Rf]
1.00
0.0
10.0
20.0
30.0
40.0
50.0
[mm]
70.0

BIO CHEMICAL ANALYSIS

BIO-CHEMICAL ANALYSIS OF TRIAL MEDICINE

Preparation of Sodium Carbonate extract:

2 gm of the sample drug is mixed 5 gm of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No	EXPERIMENT	OBSERVATION	INFERENCE
I	**TEST FOR ACID RADICALS**		
1a	**Test for Sulphate** 2 ml of the above prepared extractis taken in a test tube. To this add2ml of 4% Ammonium oxalatesolution.	Absence of White Precipitate	Absent
b	2ml of extract is added with 2ml of dilute hydrochloric acid until the effervescence ceases off. Then2ml barium chloride solution isadded.	Absence of White Precipitate	Absent
2	**Test for Chloride:** 2ml of extract is added with dilutenitric acid till the effervescenceceases. Then 2ml of silver nitratesolution is added.	Absence of white precipitate	Absent
3	**Test for Phosphate** 2ml of the extract is treatedwith 2 ml of Ammoniummolybdate solution and 2ml ofconcentrated nitric acid.	Absence of Yellow precipitate	Absent
4	**Test for Carbonate:**	Absence of white	Absent

	2ml of the extract is treatedwith 2ml of magnesiumsulphate solution.	precipitate	
5	**Test for Sulphide:** 1 gm of the substance is treated with 2ml of concentratedHcl.	Absence of Rotten egg smelling	Absent
6	**Test for Nitrate:** 1gm of the substance is heatedwith copper turnings andconcentrated sulphuric acidand viewed the test tubevertically down.	Absence of reddish brown gas.	Absent
7a	**Test for Fluoride and oxalate** 2ml of the extract is added with2ml of dilute acetic acid and2ml of calcium chloridesolution and heated.	Absence of white precipitate	Absent
b	5 drops of clear solution isadded with 2ml of diluted sulphuric acid and slightlywarmed to this, 1 ml of dilutepotassium permanganate solution is added.	Absence of Discolourisation	Absent
8	**Test for Nitrite** 3 drops of the extract is placedon a filter paper. On that, 2 drops of Acetic Acid and 2drops of Benzidine solution isplaced.	Absence of yellowish red colour	Absent
9	**Test for Borate** 2 pinches of the substance ismade into paste by usingSulphuric acid and Alcohol(95%) and introduced	Absence of Green tinged flame	Absent

	into theblue flame.		
II	**TEST FOR BASIC RADICALS**		
10	**Test for lead** 2 ml of the extract is addedwith 2 ml of Potassium iodidesolution.	Absence of Yellow precipitate	Absent
11a	**Test for Copper** One pinch of substance ismade into paste withconcentrated Hydrochloric acidin a watch glass and introducedinto the non luminous part ofthe flame.	Absence of Bluish green coloured flame.	Absent
b	2ml of the extract is added withexcess of Ammonia solution	Absence of deep blue	Absent
12	**Test for Aluminium** To the 2 ml of extract. SodiumHydroxide solution is added indrops to excess	Absence of White Precipitate.	Absent
13a	**Test for Iron** To the 2 ml of extract, 2 ml ofAmmonium ThiocyanateSolution is added.	Presence of Blood red colour	Present
b	To the 2 ml of extract, 2 ml ofAmmonium Thiocyanatesolution and 2 ml ofconcentrated Nitric Acid isadded.	Blood red colour obtained	Present
14	**Test for Zinc** To the 2 ml of extract SodiumHydroxide solution is added indrops to excess.	Absence of White Precipitate.	Absent

15	**Test for Calcium** 2 ml of the extract is addedwith 2 ml of 4% AmmoniumOxalate solution.	Presence of White Precipitate.	Present
16	**Test for Magnesium** 2ml of extract, SodiumHydroxide solution is added indrops to excess.	Absence of White Precipitate.	Absent
17	**Test for Ammonium** 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	Absence of Reddish brown precipitate	Absent
18	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2ml of Cobal Nitrate in 30%glacial Acetic acid.	Presence of Yellow precipitate	Present
19	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Absence of Yellow colour flame	Absent
20	**Test for Mercury** 2 ml of the extract is treatedwith 2 ml of Sodium Hydroxide solution.	Absence of yellow precipitate	Absent

21	**Test for Arsenic** 2 ml of extract is treated with 2ml of silver Nitrate solution.	Absence of Yellow precipitate	Absent
22	**Test for Starch** 2ml of extract is treated with weak iodine solution	Absence of Blue colour	Absent
23	**Test of reducing Sugar** 5ml of Benedicts qualitative solution is taken in a test tube and allowed to boil for 2minutes and added 10 drops of the extract and again boiled for2 minutes. The colour changes are noted.	Absence of Green colour	Absent
24	**Test of the alkaloids** 2ml of the extract is treated with 2ml of potassium lodide solution.	Presence of Red colour	Present
25	**Test of the proteins** 2ml of the extract is treated with 2ml of 5% NaOH,mix well and add 2 drops of copper sulphate solution.	Absence of Violet colour	Absent

RESULTS:

The given sample (Naval Kottai Chooranam) contains

Iron

Calcium

Potassium

Alkaloids

TOXICITY STUDY

Toxicity study

ACUTE ORAL TOXICITY – OECD GUIDELINES - 423

Acute toxicity study was carried out as per OECD guideline (Organization for Economic Co - operation and Development, Guideline-423

Animal : Healthy wistar albino female rat weighing 220–240 gm

Studied carried out at three female rat under fasting condition, signs of toxicity was observed for every one hour for first 24 hours and every day for about 14 days from the beginning of the study.

INTRODUCTION:

The acute toxic class method is a stepwise procedure with the use of 3 animals of a singlesex per step. Depending on the mortality and/or the moribund status of the animals, on average 2-4 steps may be necessary to allow judgment on the acute toxicity of the test substance. Morbid animals or animals obviously in pain or showing signs of severe and enduring distress shall be humanely killed, and are considered in the interpretation of the test results in the same way as animals that died on test. The method allows for the determination of an LD50 value only when at least two doses result in mortality higherthan 0% and lower than 100%.

PRINCIPLE:

It is the principle of the test that based on a stepwise procedure with the use of a minimum number of animals per step, sufficient information is obtained on the acute toxicity of the test substance to enable its classification. The substance is administered orally to a group of experimental animals at one of the defined doses. The substance is tested using a stepwise procedure, each step using three animals of a single sex. Absence or presence of compound-related mortality of the animals dosed at one step will determine the next step, i.e.; − no further testing is needed − dosing of three additional animals with the same dose − dosing of three additional animals at the next

higher or the next lower dose level. The method will enable a judgment with respect to classifying the test substance to one of a series of toxicity classes.

METHODOLOGY

Selection of animal species:

The preferred rodent species is rat, although other rodent species may be used. Healthy young adult animals of commonly used laboratory strain Swiss albino is used . Females should be nulliparous and non-pregnant. Each animal at the commencement of its dosing should be between 8 and 12 weeks old and its weight should fall in an interval within±20 % of the mean weight of the animals.

Housing and feeding conditions:

The temperature in the experimental animal room should be 22ºC (+3ºC). Although the relative humidity should be at least 30% and preferably not exceed 70% other than during room cleaning the aim should be 50-60%. Lighting should be artificial, the sequence being 12 hrs light, 12 hrs dark. For feeding, conventional laboratory diets may be used with an unlimited supply of drinking water. Animals may be grouped and tagged by dose, but the number of animals per cage must not interfere with clear observations of each animal.

Preparation of animals:

The animals are randomly selected, marked to permit individual identification, and kept in their cages for at least 7 days prior to dosing to allow for acclimatization to the laboratory conditions.

Observation done:

Group	Day
Body weight	Normal
Assessments of posture	Normal
Signs of Convulsion Limb paralysis	Absence of sign (-)
Body tone	Normal
Lacrimation	Absence
Salivation	Absence
Change in skin color	No significant colour change
Piloerection	Normal
Defecation	Normal
Sensitivity response	Normal
Locomotion	Normal
Muscle gripness	Normal
Rearing	Mild
Urination	Normal

Dose mg/kg	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**
2000	+	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-

1.*Alertness 2.Aggressiveness 3. Pile erection 4. Grooming 5.Gripping 6. Touch Response 7. Decreased Motor Activity 8.Tremors 9 Convulsions 10. Muscle Spasm 11. Catatonia 12.Musclerelaxant 13.Hypnosis 14.Analgesia115.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19 Respiration 20. Mortality*

Acute toxicity:

In the acute toxicity study, the rats were treated with different concentration of naval kottaichooranam from the range of 5mg/kgto 2000mg/kg which did not produce signs of toxicity, behavioral changes, and mortality in the test groups as compared to the controls when observed during 14 days of the acute toxicity experimental period. These results showed that a single oral dose of the extract showed no mortality of these rats even under higher dosage levels indicating the high margin of safety of this extract. In acute toxicity test the naval kottaichooranam was found to be non toxic at the dose level of 2000mg/ kg body weight.

SUB-ACUTE TOXICITY

The dose selected for the sub acute toxicity study was 100mg, 200mg/kg of Naval kottaiChooranam. All the animals were free of intoxicating signs throughout the dosing period of 28 days. No physical changes were observed throughout the dosing period. No mortality was observed during the whole experiment. No abnormal deviations were observed. No significant changes were observed in the values of different parameters studied when compared with controls and values obtained were within normal biological and laboratory limits. The weights of organs recorded did not show any significant differences in the treatment and the control group indicating that Naval kottaiChooranamwas not toxic to kidney, liver and spleen There was no significant changes were observed in hemoglobin (Hb), red blood cell (RBC), white blood cell (WBC), packed cell volume (PCV), Erythrocyte sedimentation rate (ESR) in all the treated groups as compared to respective control groups.

SUB ACUTE REPORTS

Naval kottai chooranam (100mg/kg)

HAEMOTOLOGY

CBC

WBC	:	5,300 cells/cumm
Differential Count		
NEUTROPHILLS	:	10%
LYMPHOCYTES	:	89 %
EOSINOPHILS	:	01 %
MONOCYTES	:	00 %
RBC	:	9.37millions/cumm
HB	:	16.6gms%
PCV	:	50.4 %

MCV : 53.8fL

MCH : 17.7pg

MCHC : 32.9 Grams/dl

PLATELET : 7.4 Lakhs/cumm

BIOCHEMISTRY

Blood sugar : 92 mg/dl

BUN : 32.1 mg/dl

Creatinine : 0.9 mg/dl

SGOT : 90 U/L

SGPT : 78 U/L

ALP : 161 U/L

T.Protein : 6.7 grams/dl

Albumin : 3.5 grams/dl

LIPID PROFILE

T. Cholesterol : 112 mg/dl

Triglycerides : 78 mg/dl

HDL : 28 mg/dl

LDL : 68.4 mg/dl

VLDL : 15.6 mg/dl

Ratio 1(T.CHO/HDL) : 4.0

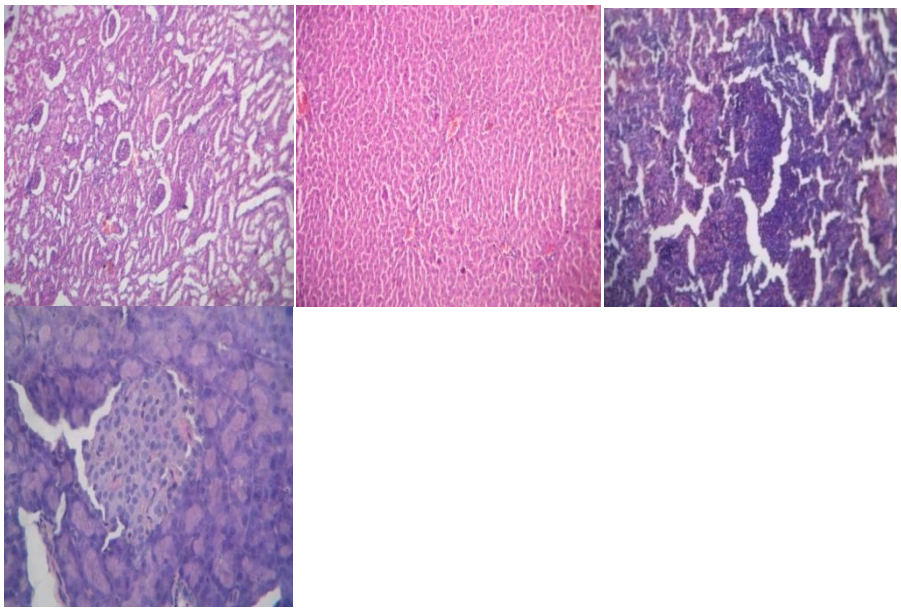
Ratio 2(LDL/HDL) : 2.44

HISTOPATHOLOGICAL REPORT

Naval kottai chooranam

(100mg/kg)

Kidney Liver Spleen pancreas



Naval kottai chooranam

(200mg/kg)

HAEMOTOLOGY

CBC

WBC : 8,700 cells/cumm

Differential Count

NEUTROPHILLS : 15%

LYMPHOCYTES : 84 %

EOSINOPHILS : 01 %

MONOCYTES : 00 %

RBC : 7.08millions/cumm

HB : 14.5gms%

PCV : 44.7 %

MCV : 63.1fL

MCH : 20.5pg

MCHC : 32.4 Grams/dl

PLATELET : 6.53 Lakhs/cumm

BIOCHEMISTRY

Blood sugar : 90 mg/dl

BUN : 42.5 mg/dl

Creatinine : 0.7 mg/dl

SGOT : 90 U/L

SGPT : 60 U/L

ALP : 101 U/L

T.Protein : 7.2 grams/dl

Albumin : 3.5 grams/dl

LIPID PROFILE

T. Cholesterol : 101 mg/dl

Triglycerides : 67 mg/dl

HDL : 24 mg/dl

LDL : 63.6 mg/dl

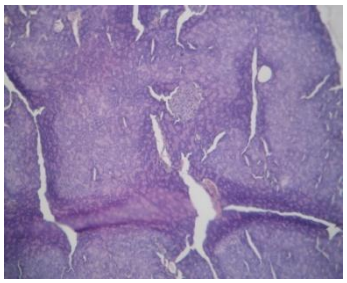
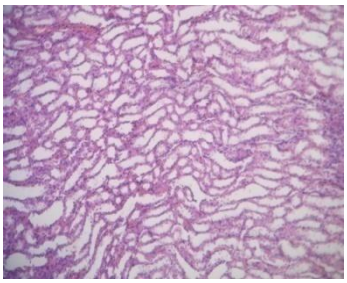
VLDL : 13.4 mg/dl

Ratio 1(T.CHO/HDL) : 4.21

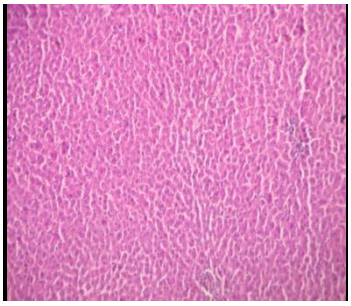
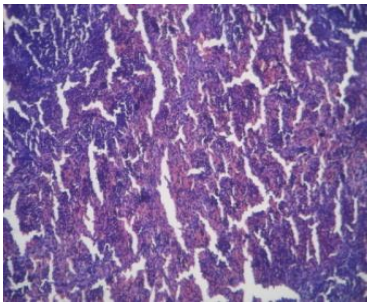
Ratio 2(LDL/HDL) : 2.65

Histopathology

Pancreas Kidney

Liver Spleen

PHARMACOLOGICAL STUDY

PHARMACOLOGICAL ACTIVITY

ANTI DIABETIC ACTIVITY

Experimental Design:

Diabetes was induced using Streptozotocin (100 mg/kg). Only Streptozotocinised animals were used for further studies. Animals were fasted for 18 h before the experiment and divided into 5 groups (6 animals in each group).

Groups	**Treatment**
Group I	Normal Control
Group II	Diabetic control- Streptozotocin (100 mg/kg)
Group III	Diabetic control- glibenclamide (5 mg/kg)
Group IV	Diabetic control- Naval kottaichooram 200mg/kg
Group V	Diabetic control- Naval kottaichooram 400mg/kg

Effects on Blood Glucose Levels

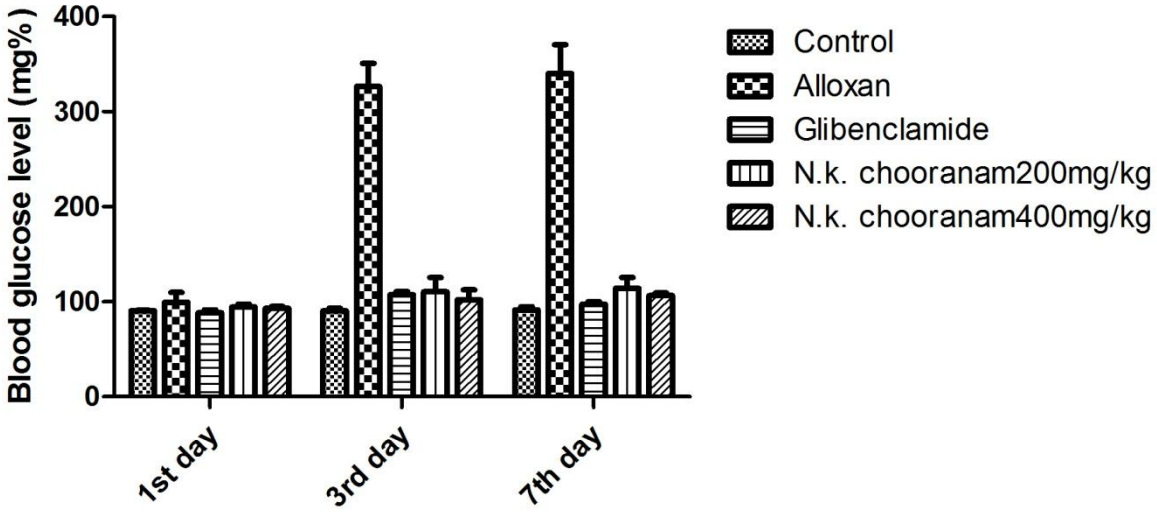
Diabetes was induced by the intravenous administration of streptozotocin (100 mg/kg)[1, 2]after anaesthesia with ethyl ether. Forty-eight hours later, the blood (1 mL) was collected from the orbital sinus into tubes and immediately usedfor the determination of glucose. Only animals that presented with glycaemic levels equal to or above 200 mg/dL were submitted to treatments,which consisted of a daily administration of naval kottaichooranam for 7 days. The oral treatments (by gavage) of all groups were carried out at the same time (in the morning) used. One hour after the lastadministration, the blood was collected again for blood glucose measurements using a glucometer

References:

Effect of Naval kottaichooranam on plasma glucose Levels on rats

Groups	Blood Glucose Levels (mg %)		
	1st day	3rd day	7th day
Control	90.33±0.88	90.66±2.40	91.33±3.52
Alloxan Control	99.66±10.17	326.66±24.03 ***	340.00±30.55 ***
Glibenclamide 5 mg/kg	88.68±2.90	107.33±3.52	97.33±2.90
Naval kottaichooranam (200mg/kg)	94.66±2.90	110.33±15.30	114.00±11.37
Naval kottaichooranam (400mg/kg)	93.00±2.08	102.00±10.39	106.66±2.40

N=6 ;Statistical analysis one way ANOVA followed by Dunnett t-test. ***P<0.01 as compared with day1.

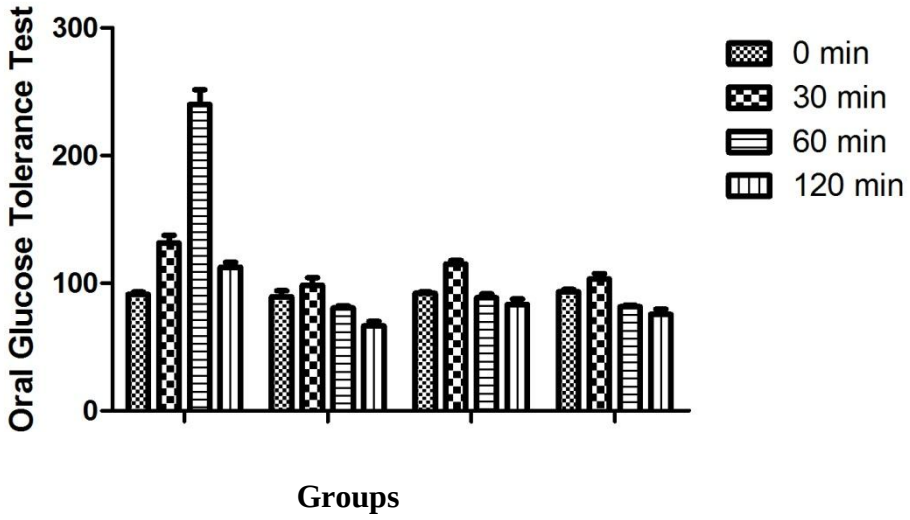


Oral glucose tolerance test:

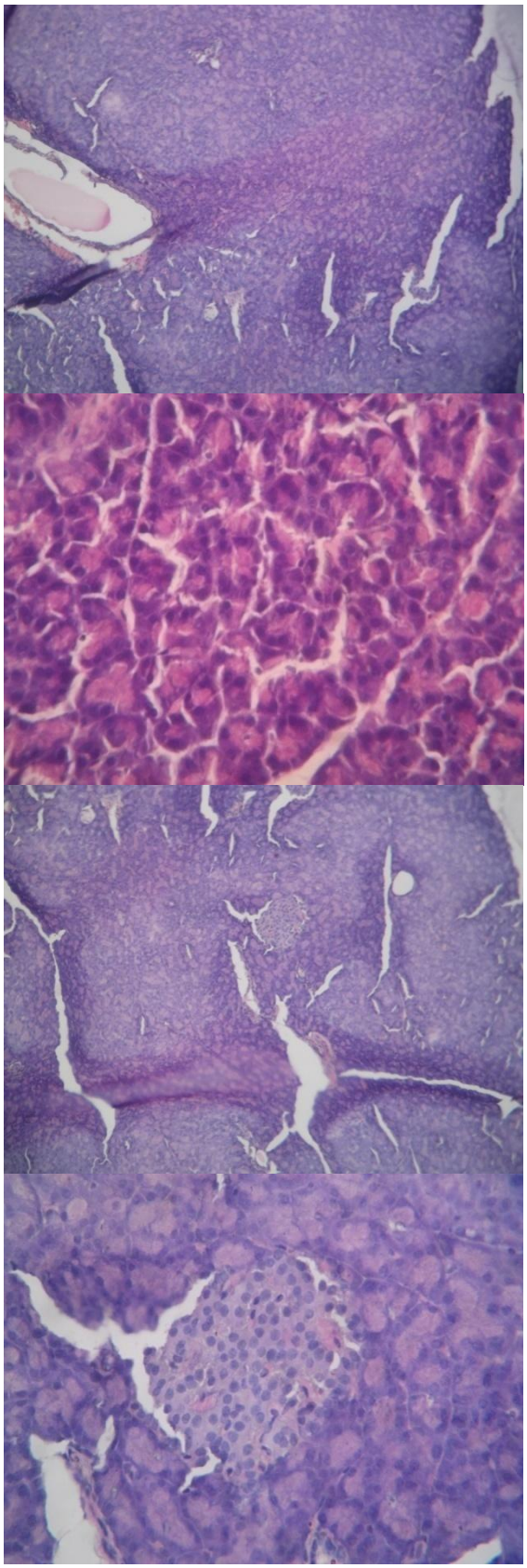
Oral glucose tolerance test (OGTT) was performed in overnighted fasted normal rats as per reported method. The blood glucose levels were determined by using glucometer by using strips [3]

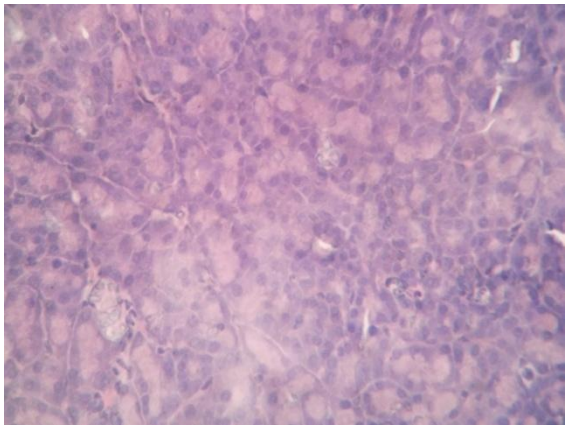
Groups	Oral Glucose Tolerance Test			
	O min	30 min	60 min	120 min
Control	90.46± 2.48	130.16± 12.14	240.56± 2.68	120. 14± 3.78
Glibenclamide	92.09± 5.65	108.56± 5.78	79.67± 4.17	64.56± 4.89
Naval kottaiChooranam 200 mg/kg	92.56± 2.48	115.16± 2.14	88.14± 7.78	86.14± 8.98
Naval kottaiChooranam 400 mg/kg	94.16± 7.78	108.14± 3.14	80.68± 3.14	76.14± 4.56

N=6 ;Statistical analysis one way ANOVA followed by Dunnett t-test. ***P<0.01



Histopathological slides of pancreas





1- Normal rats
2- Streptozotocin induced rats
3- Glibenclamide 5 mg/kg
4- Naval kottaichooranam (200mg/kg)
5- Naval kottaichooranam (400mg/kg)

Interpretation :

- Group I- Normal Saline treated rats showed using with Haematoxylin & Eosin stained section shows pancreas with normal islets and acini
- Group II- Streptozotocin treated rats showed damaged and atrophic islet with acini.
- Group III- Streptozotocin + Glibenclamide rats showed preserved and regenerated cells of islet with acini
- Group IV- Streptozotocin + N. KottaiChooranam200mg/kg showed small damaged of pancreatic islet cells.
- Group V- Streptozotocin+ N. KottaiChooranam 400mg/kg showed recovered of pancreatic islet cells.

References:

1. Vogel GH. Drug Discovery and Evaluation, 2nd ed. 2002. p. 950.

2. Onunkwo GC, Akah PA, Udeala OK. Studies on B. ferruginea leaves (I), Stability and hypoglycemic actions of the leaf extract tablets. Phytother Res 1996; 10:418-20.

3. Gokce G HaznedarogluMZ . Evaluation of antidiabetic, antioxidant and vasoprotective effects of Posidoniaoceanica extract. J Ethnopharmacol 2008; 115: 122-130.

BIO STATISTICAL ANALYSIS

CLINICAL PROGNOSIS

Treatment for Madhumegam:

The most popular non parametric statistical tool, namely, McNemar Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

S. No	Signs & Symptoms	Before Treatment	After Treatment
		n%	n%
1.	Polyuria	40(100)	7(17.5)**
2.	Polyphagia	40(100)	5(12.5)**
3.	Polydipsia	40(100)	4(10)**
4.	Pruritis vulvae	4(10)	0(0)*
5.	Itching all over the body	10(25)	0(0)**
6.	Pain all over the body	34(85)	7(17.5)**
7.	Dryness of mouth and throat	40(100)	0(0)**
8.	Constipation	8(20)	0(0)**
9.	Emaciation	4(10)	3(7.5)
10.	Peripheral neuritis	10(25)	4(10)*
11.	Disturbed sleep	40(100)	4(10) **

McNemat test, C.I: 95%, *P<0.05; **P<0.01

Software: spss17 version

Number of cases: 40

Inference:

Since the p value is significant in signs and symptoms except emaciation.So there is significant reducing of signs & symptoms except emaciation among the patients for the treatment of Madhumegam.Hence it is concluded that the treatment was effective and **significant.**

Effect of Naval kottaichooranam on Fasting Blood Sugar level in Madhumegam cases

S. no	Fasting blood sugar level in mg	
	Before Treatment	**After Treatment**
1.	166	116
2.	95	95
3.	162	114
4.	139	96
5.	136	105
6.	127	95
7.	163	109
8.	168	110
9.	199	180
10.	139	90
11.	151	110
12.	158	110
13.	190	140
14.	136	96
15.	136	98
16.	152	102
17.	189	100
18.	109	80
19.	104	90
20.	126	94
21.	181	98
22.	96	80
23.	117	100
24.	120	105
25.	157	110
26.	110	100
27.	139	109

28.	168	140
29.	140	110
30.	120	90
31.	133	100
32.	162	120
33.	198	120
34.	131	97
35.	140	100
36.	170	120
37.	158	108
38.	142	110
39.	128	84
40.	130	110

Software: spss17 version

Variables: Fasting Blood Sugar Level (mg) – before treatment, after treatment

Number of cases: 40

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.715

Before and after treatment mean difference ±SEM: 38.60 ± 2.95

P Value (2 tailed): $p<0.001$.

Inference:

Since the p value is significant ($p<0.001$). The hypothesis is not accepted. So there is significant reducing of Fasting blood sugar level (mg) among the patients for the treatment of Madhumegam. Hence it is concluded that the treatment was effective and significant.

Effect of Naval kottaichooranam on Postprandial blood Sugarlevel in Madhumegam cases

S. no	Postprandial blood sugar level in mg	
	Before Treatment	**After Treatment**
1.	262	240
2.	276	180
3.	258	182
4.	299	140
5.	280	140
6.	172	130
7.	298	138
8.	242	160
9.	289	249
10.	199	142
11.	187	140
12.	256	176
13.	272	191
14.	190	126
15.	190	142
16.	241	162
17.	289	180
18.	142	120
19.	213	150
20.	212	138
21.	288	142
22.	168	140
23.	170	120
24.	180	150
25.	263	160
26.	160	130
27.	181	130
28.	299	180

29.	256	160
30.	168	142
31.	249	160
32.	258	160
33.	311	200
34.	169	131
35.	210	160
36.	200	150
37.	195	160
38.	194	140
39.	278	132
40.	210	160

Software: spss17 version

Variables: Postprandial Blood Sugar Level (mg) – before treatment, after treatment

Number of cases: 40

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.585

Before and after treatment mean difference ±SEM: 73.52 ± 6.23

P Value (2 tailed): $p<0.001$.

Inference:

Since the p value is significant ($p<0.001$). The hypothesis is not accepted. So there is significant reducing of postprandial blood sugar level (mg) among the patients for the treatment of Madhumegam.Hence it is concluded that the treatment was effective and significant.

Effect of Naval kottaichooranam on HbA1C level in Madhumegam cases

S. no	HbA1C	
	Before Treatment	After Treatment
1.	8.9	7.8
2.	11.2	7.2
3.	8.3	6.9
4.	9.6	6.2
5.	9.5	6.4
6.	6	5.8
7.	10.2	8.2
8.	10.4	8.1
9.	11.6	8.8
10.	8	6.5
11.	7.7	6.2
12.	9.1	7.6
13.	11.2	7.8
14.	8.3	6.9
15.	6.9	6.1
16.	9.2	7.1
17.	11	7.3
18.	6.5	6.1
19.	6.7	6.2
20.	9.5	4.9
21.	9.8	7.1
22.	6.4	6.1
23.	6.7	6.2
24.	6.5	6.1
25.	8.21	6.2
26.	6.7	6.1
27.	8.1	6.1
28.	13.1	8.5
29.	8.9	6.5

30.	6.8	6.1
31.	7.6	6.4
32.	10.1	7.2
33.	10.3	7.2
34.	7.2	6.2
35.	8.3	7.1
36.	8.1	7
37.	8.1	6.7
38.	8.3	7
39.	9.1	6.8
40.	7.9	6.5

Software: spss17 version

Variables: HbA1C Level – before treatment, after treatment

Number of cases: 40

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.741

Before and after treatment mean difference ±SEM: 1.87 ± 0.19

P Value (2 tailed): $p<0.001$.

Inference:

Since the p value is significant ($p<0.001$). The hypothesis isnot accepted. So there is significant reducing of HbA1C level among the patients for the treatment of Madhumegam.Hence it is concluded that the treatment was effective and significant.

CONSENT FORM

CONSENT FORM

I certify that I have disclosed all the details about the study in the terms readily understood by the patient.

DATE: **SIGNATURE**

NAME

CONSENT BY THE PATIENT

I have been informed to my satisfaction by the attending physician the purpose of the clinical trial and the nature of the drug treatment and follow up including the lab investigations to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of the trial without having to give reasons for doing so.

I am exercising my free power of choice, and hereby give my consent to be included as a subject in the clinical trial of **NAVAL KOTTAI CHOORANAM** for the treatment of **MADHUMEGAM.**

DATE: **SIGNATURE**

NAME

நோயாளியின் ஒப்புதல் படிவம்

னிரு---------- ஆகிய நான் ---- வயது----------

---------- என் சுய நினைவுடன் எழுதிக்கொடுக்கும் ஒப்புதல் படிவ□.

நான் **மதுமேக□ (பு□□ளி)** என்னும் நோயால்பாதிக்கப்பட்டுசென்னைஅரசுசித்தமருத்துவகல்லூரியில்(இடம்: அ□ஞ□ அண்ணாமருத்துவமனை, அரும்பாக்கம், சென்னை– 106) நடமீத□படு□ சித்தமருத்துவஆராய்ச்சி மூலம் சிகிச்சைபெற என் முழு சம்மதத்தையும் தெரிவித்துக்கொள்கிறேன்.

இந்தஆராய்ச்சியின் நோக்கம், மருத்துவம் செய்யும் முறைதொடர் கண்காணிப்புமற்றும் என் உடல்நலம் குறித்த மருத்துவபரிசோதனைகளை பற்றி □ □வான□ ள□க□ எனக்குமருத்துவம் செய்யும் மருத்துவர் மூலம் தெளிவுபடுத்தப்பட்டுள்ளது. இந்தஆராய்ச்சியில்பங்குகொள்ளும் என் சம்மதத்திற்குயாருடையநிர்பந்தமும் காரணமுமில்லைஎன்பதைதெரிவித்துக்கொள்கிறேன்.

□□படி□கு,

பெய□:

முகவ□:

நாள்:

CASE SHEET

PROFORMA

CASE SHEET PROFORMA

POST GRADUATE DEPARTMENT - BRANCH-I MARUTHUVAM

GOVT. SIDDHA MEDICAL COLLEGE & ANNA HOSPITAL, CHENNAI-106.

CASE SHEET PROFORMA FOR "MADHUMEGAM"

Ward No.	:	Nationality	:
I.P. No	:	Religion	:
Bed No	:	Occupation	:
Name	:	Income	:
Age	:	Date Of	
Sex	:	Admission	:
Permanent Address:		Date Of	
		Discharge	:
		Diagnosis	:
Temporary Address:		Medical Officer	
Govt. Siddha Medical College &			
Anna Hospital, Chennai – 106.			

Complaints And Duration :

History Of Present Illness :

History Of Past Illness :

Personal History & Habits :

Family History :

GENERAL EXAMINATION:

1. Body built
2. Consciousness
3. Nourishment
4. Decubitus
5. Anaemia
6. Jaundice
7. Cyanosis
8. Clubbing
9. Lymphadenopathy
10. Oedema
11. Jugular venous pulsations
12. Enlarged vein
13. Miscellaneous

VITAL SIGNS:

14. Pulse rate
15. Temperature
16. Respiratory rate
17. Heart Rate
18. Blood Pressure.

SIDDHA ASPECTS

NILAM (PLACES)

1. Kurinchi (Moutains and their adjoining Areas)
2. Mullai (Forest and their adjoining Areas)
3. Marudham (Fertile and their adjoining Areas)
4. Neithal (Sea and their adjoining Areas)
5. Paalai (Desert and their adjoining Areas)

PARUVA KAALAM (SEASONS)

1. KaarKaalam (Aavani-Puratasi) Aug-sept.
2. KoothirKaalam (Iypasi-Karthigai) Oct-Nov.
3. MunpaniKaalam (Maargazhi-Thai) Dec-Jan.
4. ElavenilKaalam (Chithirai-Vaikasi) Apr-May
5. MudhuvenilKaalam (Aani-Aadi) Jun-Jul

YAAKAI (UDAL)

1. Valiudal
2. Azhaludal
3. IyyaUdal
4. KalappuUdal

UTKAYAM (Athakkayam)

1. Puyam
2. Sayam
3. Kall
4. Patham

GUNAM

1. SathuvaGunam
2. RajoGunam
3. ThamoGunam

IYAMPORIGAL (SENSORY ORGANS)

1. Mei - Unarthal
2. Vaai - Suvaiththal
3. Kan - Parththal
4. Mooku - Mugarthal
5. Sevi - Kettal

KANMENTHIRIYAM / KANMAVIDAYAM

1. Kai - Koduththal
2. Kaal - Nadaththal
3. Vaai - Pesal
4. Eruvai - MalamKazhithal
5. Karuvai – Aananthithal

KOSAM

1. AnnamayaKosam
   (YeluudalThaathukkal)

2. PranamayaKosam
   (Pranan + Kanmenthiriyam)

3. ManomayaKosam
   (Manam + Gnanethiriyam)

4. GnanamayaKosam
   (Puththi + Gnanenthiriyam)

5. AnanthamayaKosam
   (Pranan + Suzhuthi)

PIRA URUPUKALIN NILAI:

1. Irudhayam
2. Puppusam
3. Eraippai
4. Kalleral
5. Manneeral
6. Kudal
7. Siruneeragam
8. Karuppai
9. Moolai

UYIR THATHUKKAL:

Vatham

1.Pranan

2. Abanan

3.Viyanan

4.Udhanan

5.Samanan

6.Naagan

7.Koorman

8.Kirukaran

9. Devadathan

10.Thanenjeyan

PITHAM:

1. Anal Pitham
2. RanjagaPitham
3. SaadhagaPitham
4. AalosagaPitham
5. PrasagaPitham

KAPHAM:

1. Avalambagam
2. Kledagam
3. Podhagam
4. Tharpagam
5. Santhigam

UDAL THATHUKKAL:

1. Saaram
2. Senneer
3. Oon
4. Kozhuppu
5. Enbu
6. Moolai
7. Sukkilam / Suronitham

EnvagaiThervu:

1. Naa - Niram

Thanmai

Pulan

2. Niram

3. Mozhi - Thazhnthaoli
Urathaoli

4. Vizhi - Niram

5. Sparisam - Thanmai
Pulan

6. Malam - Niram
Erugal / Elagal
Manam
Nurai

7. Moothiram

a. Neerkuri - Niram, Manam, Edai, Nurai, Enjal

b. Neikuri

8. Naadi - ThaniNaadi
KalappuNaadi
MukkutraNaadi
ThondhaNaadi

Systemic Examination:

1.Gastro intestinal system

2.Cardio Vascular System

3.Respiratory system

4.Central Nervous System

5.Genito-Urinary System

Signs and Symptoms:

S.no	Clinical Features	Before treatment	After treatment(Weeks)						
			I	II	III	IV	V	VI	VII
1.	Polyuria								
2.	Polyphagia								
3.	Polydipsia								
4.	Balanitis / Pruritis vulva								
5.	Itching all over body								
6.	Pain all over body								
7.	Dryness of the mouth and throat								
8.	Constipation								
9.	Skin infection								
10	Emaciation								
11	Dry skin								
12	Peripheral Neuritis								
13	Ulcer in the foot								
14	Diabetic cataract								

Laboratory Investigation:

I. Blood
- TC
- DC
- ESR
- Hb %
- Sugar -Fasting
  - Post prandial
- Glycosylated Haemoglobin (HbAIC)
- Blood urea
- Serum cholesterol

2. Urine
- Albumin
- Sugar
- Deposits.

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CASE SUMMARY:

FINAL DIAGNOSIS:

MEDICINE:

NAVAL KOTTAI CHOORANAM- 1g BD with warm water

MEDICAL ADVICE **HOD**

BIBLIOGRAPHY

BIBLIOGRAPHY

1) **1**. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition-2014 pg.120

29. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition- 2014 pg.97

30. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition-2014 pg.269

32.Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition-2014 pg.282

33. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition-2014 pg.282

34. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition- 2014 pg.298

35. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition-2014 pg.299

51. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition-2014 pg.218

52. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition-2014 pg.218

53. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition-2014 pg.219

54. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-1, 6th edition-2014 pg.219

(2) **2**. Dr. R. Thiagarajan LIM , SirappuMaruthuvam, 3rd edition pg.4

(**3)** **3.** A Review on anti diabetic herbs in siddha medicine IJPA 3(3) 2014 1-8

(4) **4**. Dr. R. Thiagarajan, Theraiyarvagadam, 1st edition 1975 pg.42

19. Dr. R. Thiagarajan, Theraiyarvagadam, 1st edition 1975 pg.42

24. Dr. R. Thiagarajan, Theraiyarvagadam, 1st edition 1975 pg.42

50. Dr. R. Thiagarajan , Theraiyarvagadam , 1st edition 1975 pg.42

(5) 5. Dr. R. Thiagarajan, Agathiyarkanmakandam 300, Arulmigudandayudhapani swami thirukovilveliettukuzhu ,2nd edition 1976 pg.1

(6) 6.Dr.M.Varadharasanar, Marunthu athigaram-95 thirukuralthelivurai edition 1998 page no 193

(7) 7. MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.149

12. MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.146

15. MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.146

16. MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.146

21.MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.155

23. MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.145

25. MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.146

26.MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.148

27. MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.151

28. MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.181

49.MozhipeyarpuDr.Anaivarianandan, Yugivaidyachinthamani, 2nd edition 2005, pg.156

(8) **8.**Theraiyarmahakarisaluraiasiriyar , Dr. R. Thiagarajan, 1st edition 1974 , pg 133

(9) **9.**T.V.Sambasivampillai dictionary volume 4 , pg.1831

(10) **10**. K.Vasudevashastri , S.Venkatrajan , Sarabendrar mega nivaranabodhiniennumneerizhivumaruthuvam, saraswathimahalnoolagam , Thanjavur , 3rd edition 2005 , pg.1

(11) **11.**Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-2, 5th edition-2010 pg.458

13.Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-2, 5th edition-2010 pg.496

14. Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-2, 5th edition- 2010 pg.496

18.Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-2, 5th edition-2010 pg.458

20.Dr. M .Shanmugavelu H.B.I.M, Noinadalnoimudalnadal part-2, 5th edition-2010 pg.460

(12) **17**.S.P.Ramachandran , Agathiyarvaidyakaviyam 1500 , Thamarainoolagam , Chennai – 26, 1st edition july 1992 , pg.9

(13) **22.**Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.494

36.Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.511

38. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.511

39. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.506

40. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.506

41. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.507

42. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.507

43. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.507

44. Dr.K.N.Kuppusamymudaliar, siddha maruthuvam, 7th edition 207, pg.507

45. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.507

46. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.507

47. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.507

48. Dr.K.N.Kuppusamymudaliar , siddha maruthuvam, 7th edition 207, pg.507

(14) **31.**Dr.K.S.Uthamarayan , Thotrakiramaaraichium siddha maruthuva varalarum,3rd edition 2006,pg.71

(15) **37.**Kalathur Kandasamymudaliar, pathinensitharnaadisasthiram , editon 2012 , pg.112

(16) **55.**Sir Stanley Davidson , Davidson's principle and practice of medicine , 22nd edition, pg.800

(17) **56**.Dr.B.D.Chaurashia , Human anatomy volume 2 , 4th edition 2004 , pg.284 and 285

(18) **57**.Dr.mohan , A guide to foot care, 1st edition 2006, pg.30

(19) **58**.Anna hospital pharmacopoeia , directorate of Indian medicine and homeopathy , pg.11

(20) **59**.Dr.K.S.Murugesa mudaliar , siddha matericamedica , edition 2008,pg.410

60. Dr.K.S.Murugesamudaliar , siddha matericamedica , edition 2008,pg.621

62.Dr.K.S.Murugesamudaliar , siddha matericamedica , edition 2008,pg.369

63.Dr.K.S.Murugesamudaliar , siddha matericamedica , edition 2008,pg.369

64. Dr.K.S.Murugesamudaliar , siddha matericamedica , edition 2008,pg.572

66. Dr.K.S.Murugesamudaliar , siddha matericamedica , edition 2008,pg.84

68.Dr.K.S.Murugesa mudaliar , siddha matericamedica , edition 2008,pg.68

21) **61**.Bakhru H.K. Herbs that heal, orient paper books , a division of vision books private ltd. New Delhi , 11th edition, pg.112-115

22) **63.**Grijeshkumar mall, Pankajkishormishra, Veeruprakash, Anti hypo lipidemic activity of Gymnemasylvestre in alloxon induced diabetic rats, global journal of biotechnology and biochemistry , 2009, pg.37 – 42

23) **65**.Rahul gupta , Anandsaxena , hypoglycaemic and anti hyperglycaemic activities of syzygiumcumini seeds in streptozotocin induced diabetic , 2011; 1(3) , 270-272.

24) **67**.Patil MB, Arunapai , Antidiabetic activity of cassia auriculata in alloxan induced diabetes mellitus in rat, international journal of natural product science,2012; Spl issue 1;135.